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ABSTRACT

Noting that children may be affected by environmental contaminants in ways quite different from the way adults are affected, this report is the second on trends in measures reflecting environmental factors that may affect the U.S. children's health and well-being. A list of measures and key findings begins the report, followed by five main sections. Several measures throughout the report are analyzed by children's race/ethnicity and family income. Section 1 presents measures showing incidence of exposure to critical concentrations of contaminants in outdoor air, indoor air, water, food, and soil. Section 2 presents measures of lead, mercury, and cotinine (marker of environmental tobacco smoke exposure) measured in the bodies of children and women. Section 3 presents trends in asthma, other severe respiratory illnesses, childhood cancers, and neurodevelopmental disorders such as attention deficit/ hyperactivity disorder and mental retardation. Section 4 concerns the emerging issues of mercury in fish and attention deficit/hyperactivity disorder. Section 5 presents data specific to California and Minnesota. The final section of the report discusses improvements in the measures and data sources and new measures for inclusion in future reports. Major findings presented in the report include declines throughout the 1990s in the percentage of days with unhealthy air quality,

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median blood lead levels for children under 5, and median blood levels of cotinine; and overall increases in the percentage of children with asthma. The frequency of new childhood cancer cases has been stable since 1990. Recent data indicate that .6 percent of children are diagnosed with mental retardation and 6.7 percent of children are diagnosed with attention deficit/ hyperactivity disorder. State data revealed that 32 percent of California public elementary schools have deterioration of lead-based paint, and 47 percent of Minnesota schools sprayed pesticides in classrooms. A glossary of terms completes the report. Four appendices include data tables and a list of environmental health objectives in the Energy Protection Agency's strategic plan. Each report section contains references. (KB)

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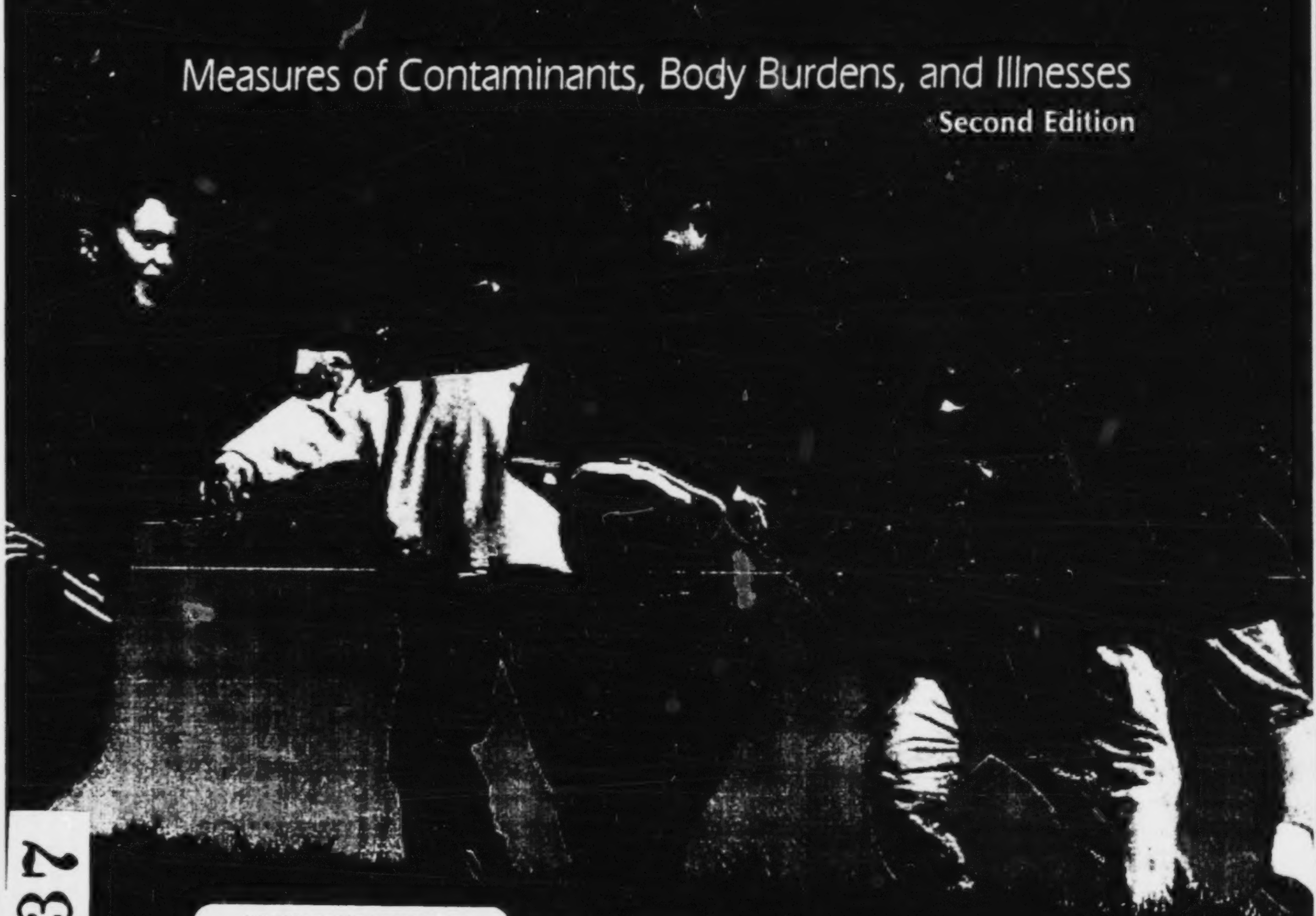
AMERICA'S CHILDREN AND THE ENVIRONMENT

Measures of Contaminants, Body Burdens, and Illnesses

Second Edition



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United States Environmental Protection Agency

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Office of Children's
Health Protection

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Foreword

I am pleased to present the U.S. Environmental Protection Agency's second edition of *America's Children and the Environment: Measures of Contaminants, Body Burdens, and Illnesses*. This report marks the progress we have made as a nation to reduce environmental risks faced by children.

The report contains good news for children including the continued decline in the number of children with elevated blood levels, a reduction in children's exposure to secondhand smoke, and decreases in exposures to air pollution and contaminants in drinking water.

Although we are encouraged by these findings, there is still much work to be done. Asthma rates are increasing, too many children continue to have elevated blood lead levels, the potential for mercury exposure in the womb is of growing concern, and there is a disproportionate impact of childhood diseases on low-income and minority children. *America's Children and the Environment* will help focus our efforts in addressing these problems and others.

Protecting children's health is an integral part of EPA's mission, and the Agency has taken great strides to improve the environment for children where they live, learn, and play, including:

- **Reducing emissions of diesel pollutants** from trucks and buses, which will help prevent hundreds of thousands of asthma attacks in children each year.
- Adopting **stringent restrictions on the use of the organophosphate pesticides** azinphos-methyl, chlorpyrifos, methyl parathion, and diazinon on food crops and around the home.
- **Taking preventive action** to reduce risks of exposure from environmental contaminants, including our work with industry to ensure playground equipment is no longer made with wood treated with arsenic-containing preservatives.
- Establishing 12 **Centers for Children's Environmental Health and Disease Prevention Research**, in partnership with the Department of Health and Human Services, to enhance scientific understanding of the relationships between environmental contaminants and children's health.
- Launching a **comprehensive schools initiative** to create healthier classrooms.
- Implementing the **Smoke-Free Home Pledge** campaign, designed to protect millions of children from the risks of secondhand tobacco smoke at home.
- Working with other federal agencies to develop and implement the **Interagency Asthma and Lead Strategies** to reduce the disproportionate impact of asthma on minority and low-income children and to eliminate childhood lead poisoning by the year 2010.
- Developing the **Clear Skies** Initiative, to reduce emissions of sulfur dioxide, nitrogen oxides, and mercury from electric utilities by approximately 70 percent, which will help reduce asthma attacks and respiratory infections.

Foreword

As we move forward, EPA is committed to monitoring the success of our children's health efforts. The *America's Children and the Environment* report, based on the best data available at this time, is an important benchmark that EPA will use to guide our future actions and measure progress. As our data and methods improve, we will work to develop increasingly reliable children's environmental health indicators that will help us in reaching our children's health goals.

I want to thank the many individuals who contributed to this report for their hard work and efforts. By monitoring trends, identifying successes, and pinpointing areas of concern, we can continue to improve the health of our children and the health of all Americans.

Christine Todd Whitman
Administrator

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About This Report

America's Children and the Environment: Measures of Contaminants, Body Burdens, and Illnesses is the U.S. Environmental Protection Agency's second report on trends in environmental factors related to the health and well-being of children in the United States.

America's Children and the Environment brings together, in one place, quantitative information from a variety of sources to show trends in levels of environmental contaminants in air, water, food, and soil; concentrations of contaminants measured in the bodies of children and women; and childhood illnesses that may be influenced by exposure to environmental contaminants.

EPA's first report, *America's Children and the Environment: A First View of Available Measures*, published in December 2000, presented the results of EPA's initial effort to collect and analyze existing, readily available data on measures relevant to children's health and the environment. This second report improves on the first edition by adding new measures for important contaminants, exposures, and childhood illnesses and by including data for additional years. The report also includes more analysis of these measures by race/ethnicity of children and family income.

What are the purposes of this report?

This report has three principal objectives. First, it presents concrete, quantifiable measures for key factors relevant to the environment and children in the United States. These measures offer a basis for understanding time trends for some factors and for further investigation of others. Second, the report can inform discussions among policymakers and the public about how to improve federal data on children and the environment. Third, *America's Children and the Environment* includes measures that can be used by policymakers and the public to track and understand the potential impacts of environmental contaminants on children's health and, ultimately, to identify and evaluate ways to minimize environmental impacts on children. The authors and sponsors hope this report will contribute to the effort to integrate the environmental health needs of children into the nation's policy agenda.

What's new in this edition of the report?

Most measures that were included in the first edition of *America's Children and the Environment* have been updated to include data for additional years. Several new measures have been added for this edition, and analyses by race/ethnicity and family income are included where possible.

What's new in the section on environmental contaminants?

- New measures of long-term exposures to outdoor air pollutants
- An improved measure of children's proximity to hazardous waste sites

What's new in the section on body burdens?

- A new measure that shows the full current distribution of blood lead levels in children ages 1-5
- A new measure of mercury in the blood of women of child-bearing age
- A new measure of cotinine (a marker of exposure to environmental tobacco smoke) in the blood of children

What's new in the section on childhood illnesses?

- New measures of respiratory diseases
- A new measure on mental retardation

A new section on emerging issues presents information about important aspects of children's environmental health for which data recently have become available. Topics covered in this section include mercury in fish, an important source of mercury exposure for people in the United States, and attention-deficit/hyperactivity disorder, which recent research suggests may be associated in part with exposure to environmental contaminants.

Also new in this report is a special features section that presents important aspects of children's environmental health for which nationally representative data are not available. The special features are based on data from single states, and include:

- Lead in paint, water, and soils in California schools
- Pesticide use in schools in Minnesota
- Birth defects trends in California

How is the report structured?

The measures in the report focus on contaminants in the environment, contaminants in the bodies of children and women, and illnesses for which there is reason to believe that environmental exposures may play a role. Measures show trends over time whenever possible.

The first part of the report presents measures reflecting trends in levels of environmental contaminants that are likely to affect children's health. These measures show the percentages of children exposed to particular levels of contaminants in air, water, food, and soil. Where data on actual environmental concentrations of contaminants are not available, the report presents surrogate measures.

The second part presents measures reflecting trends in concentrations of key contaminants measured in the bodies of children and women. These data provide direct evidence of exposures.

The third part presents measures that reflect trends in key childhood illnesses, the frequency or severity of which may be related to exposure to environmental contaminants.

The fourth part presents information about emerging issues for which data recently have become available or that are new to this report.

The fifth part presents measures for important aspects of children's environmental health based on data from single states.

Ideally, it would be informative to include measures that reflect similar environmental health concerns for children in all three of the report's main topic areas—exposure to contaminants in the environment (Part 1), concentrations of the same contaminants in the bodies of children and women (Part 2), and illnesses for which these contaminants have been found to play a role (Part 3). Although there are not sufficient data to fully accomplish this goal, relationships among some of the measures in the three sections are evident.

For example, Part 1 includes measures that reflect children's exposures to outdoor air pollutants over both the short and long term, while Part 3 includes measures for respiratory diseases, some of which are associated with air pollution. Similarly, for environmental tobacco smoke, a key pollutant of indoor air, Part 1 includes measures reflecting the frequency of smoking in homes where children live; Part 2 includes a measure for concentrations of cotinine, a marker for exposure to environmental tobacco smoke, in the blood of children; and Part 3 includes a measure on respiratory-related health effects that can in part be caused by exposure to environmental tobacco smoke. In another example, Part 1 includes a measure on the percentage of children living in counties in which air quality standards for lead were exceeded; Part 2 includes measures on concentrations of lead in the blood of children; Part 3 includes a new measure for mental retardation in children, which may be due in part to exposure to lead; and Part 5 has information from California on lead in schools.

Appendix A provides tables showing the data on which the measures were based. Appendix B describes the sources of the data used in this report and the methods for calculating the measures. Appendix C has a list of health goals relevant to the topics in this report, developed by Healthy People 2010, a collaborative effort coordinated by the U.S. Department of Health and Human Services to establish national health objectives. Appendix D lists EPA's Government Performance and Results Act goals that are related to the measures in this report. These goals are set to achieve EPA's overall objectives of clean air, clean and safe water, safe food, and the protection of America's land.

The report includes a discussion of future directions, including ways in which the existing measures could be improved and additional measures that may be included in future editions.

Why did EPA focus on measures for children?

Environmental contaminants can affect children quite differently than adults, both because children may be more highly exposed to contaminants and because they may be more vulnerable to the toxic effects of contaminants.

Children generally eat more food, drink more water, and breathe more air relative to their size than adults do, and consequently may be exposed to relatively higher amounts of contaminants. Children's normal activities, such as putting their hands in their mouths or playing on the ground, can result in exposures to contaminants that adults do not face. In addition, environmental contaminants may affect children disproportionately because their immune defenses are not fully developed and their growing organs are more easily harmed.

How were the measures in this report selected?

Three principal criteria were used to select measures for the report: 1) importance to the health of children, 2) availability of data for much or all of the United States, and 3) and sufficient quality of data to generate a reliable measure.

For environmental contaminants, five important media were identified: outdoor air, indoor air, drinking water, food, and soil. For each of these media, data available from federal environmental and health agencies were reviewed. The most informative sources that provided national coverage (or close to it) and a reasonable assurance of reliability were selected. If data about concentrations of key contaminants could be identified and were of adequate quality, they were used. If not, the best available surrogate measure was selected.

The available data for concentrations of contaminants in the bodies of children and women were reviewed, and the report presents selected contaminants for which several years of data were available or for which health impacts had been well established. These are lead, mercury, and cotinine (the latter of which reflects exposure to environmental tobacco smoke)—pollutants long recognized as having important impacts on children's health. The report presents the best available information about the concentrations of lead and cotinine in the blood of children, and about the concentrations of mercury in the blood of women of child-bearing age.

For childhood illnesses associated with environmental contaminants, the report presents measures of asthma and other respiratory conditions, childhood cancer, and neurodevelopmental disorders. The best available data to assess the frequency of these illnesses in children were selected, with measures structured to portray changes over time, where possible.

In cases where data are not available for a sufficient number of years, measures are structured as snapshots; in future editions EPA expects to have data that can be used to portray trends for those measures.

In the special features section, the report presents measures that reflect important aspects of children's environmental health for which data were not available at the national level. These were chosen based on recommendations from peer reviewers and others.

America's Children and the Environment is intended to convey information about trends in children's environmental health in the United States. The key measures presented in this report are based on the best available data to provide the most complete picture possible at this time. There are certain data limitations and assumptions in some of the measures, resulting in a degree of uncertainty for certain key measures and trends. As data and methods improve, we aim to develop increasingly reliable indicators of children's environmental health.

The *America's Children and the Environment* report, and the key measures used in the report, should not be construed as a definitive basis for planning specific policies or projects. Other technical information also will be used to inform the activities of EPA and other federal agencies concerning children's environmental health. Emerging and ongoing research will help shape these activities for years to come.

What are the sources for the data in this report?

Federal agencies provided the data for most of the measures. The data on environmental contaminants generally are from data systems maintained by EPA and by state environmental agencies. Data on contaminants in blood and on respiratory diseases and neurodevelopmental disorders are from the National Center for Health Statistics in the Centers for Disease Control and Prevention. Cancer data are from the National Cancer Institute. Population data from the Census Bureau were used to calculate the number of children potentially exposed to environmental contaminants.

Data for the special features section are from the states of California and Minnesota. The data on lead in schools are from a survey of schools in California. The data on pesticide use in schools are from a survey of Minnesota schools. The data on birth defects are from California's birth defects monitoring program.

Detailed descriptions of the data sources are in Appendix B.

What groups of children are included in this report?

Most of the measures include all children in the United States under the age of 18, representing approximately 72 million individuals based on the 2000 census. Exceptions are noted in Appendix B.

In response to suggestions from peer reviewers, the report presents (where possible) measures for groups of children of different races and ethnicities and for children living in households with various levels of income. In some cases, these breakouts by race/ethnicity and family income are shown in the graphs, while in other cases they are included in the data tables found in Appendix A.

About This Report

The report uses five categories of race or ethnicity: White non-Hispanic, Black non-Hispanic, Hispanic, American Indian/Alaska Native, and Asian or Pacific Islander. In many cases, the data were insufficient to present results for the latter two categories.

The report uses three categories of family income: 1) below the poverty level (shown in graphs and tables as < Poverty Level), 2) between the poverty level and twice the poverty level (100-200% of Poverty Level), and 3) more than twice the poverty level (> 200% of Poverty Level). "Poverty level" is defined by the federal government and is based on income thresholds that vary by family size and composition. The category of incomes between the poverty level and twice the poverty level represents households that have relatively low incomes but are not below the officially defined poverty level. This category frequently is used by the Centers for Disease Control and Prevention in its reporting of health data and was recommended by peer reviewers for use in this report.

What years are included in this report?

The report includes data for each year from 1990 through 2000 whenever possible. In many cases, data were available for only some of these years. In other cases, data available before 1990 or after 2000 were included to provide an expanded depiction of trends.

Is this report available online?

This report is available at www.epa.gov/envirohealth/children. In addition, the Web site includes links to other information on children's environmental health, additional data tables, information by state where such data are available, and references.

How does the information in *America's Children and the Environment* differ from what is proposed to be included in EPA's forthcoming *Report on the Environment*?

EPA is developing a report on the state of the environment in the United States, scheduled for publication in 2003. The *Report on the Environment* is intended to be a broad-based collection of national data depicting progress in addressing environmental problems and identifying remaining challenges. *America's Children and the Environment* focuses more specifically on data related to children and their related environmental conditions. Both *America's Children and the Environment* and the forthcoming *Report on the Environment* rely on existing national data to describe current conditions and trends.

The forthcoming *Report on the Environment* will address a broader set of environmental conditions and human health concerns. The *Report on the Environment* will be organized around five theme areas: 1) human health, 2) ecological health, 3) air, 4) water, and 5) land. *America's Children and the Environment* is organized into three main sections: 1) environmental contaminants, 2) body burdens, and 3) childhood health. A fourth section of special features presents important measures for which data are available from individual states but not for the nation.

What is the Office of Children's Health Protection at EPA?

The Office of Children's Health Protection (OCHP) supports and facilitates EPA's efforts to protect children from environmental threats. OCHP's mission is to make the protection of children's health a fundamental goal of public health and environmental protection in the United States. OCHP reviews EPA proposals for their impact on children and funds work designed to improve the protection of children from environmental hazards.

What are the Office of Policy, Economics, and Innovation and the National Center for Environmental Economics at EPA?

The Office of Policy, Economics, and Innovation develops new approaches and provides analysis to enable EPA to better address emerging environmental challenges. The office addresses cross-cutting environmental management strategies, identifies emerging issues, and serves as a catalyst for testing and institutionalizing integrative approaches to environmental protection.

Within the Office of Policy, Economics, and Innovation, EPA's National Center for Environmental Economics (NCEE) provides economic and health analysis of important environmental issues for the regulatory and policy process. NCEE also conducts research that will improve our current understanding of the impacts of environmental contaminants on public health. NCEE's staff includes specialists in air, water, solid waste, cross-media economics, and children's health risks. The center's health scientists emphasize new methods for assessing previously unidentified risks, assessing relationships between exposures and disease, and developing tools to communicate this information to the public.

Key Findings

Part 1: Environmental Contaminants

Outdoor Air Pollutants

- In 1990, approximately 23 percent of children lived in counties in which the one-hour ozone standard was exceeded on at least one day per year. In 2001, approximately 15 percent of children lived in such counties. This value fluctuated during the intervening years, ranging from 13 to 28 percent. (Page 23)
- In 1996-2001, significantly more children lived in counties that exceeded the eight-hour ozone standard than in counties that exceeded the one-hour standard. In 2001, nearly 40 percent of children lived in counties that exceeded the eight-hour standard. (Page 23)
- In 2000, approximately 27 percent of children lived in counties that exceeded the PM-2.5 particulate matter standard. In 2001, approximately 25 percent of children lived in such counties. (Page 23)
- The percentage of days that were designated as having "unhealthy" air quality (including days that were unhealthy for everyone as well as those that were unhealthy for sensitive groups) decreased between 1990 and 1999, dropping from 3 percent in 1990 to less than 1 percent in 1999. The percentage of days with "moderate" air quality remained around 20 percent between 1990 and 1999, although an upward trend is suggested by the fact that the percentage of moderate air quality days was higher in 1999 than for any other year in this analysis. (Page 25)
- In 1990, on average, children were exposed to 31.9 micrograms per cubic meter of PM-10, which represents 64 percent of the standard for the year. By 1995, the concentration had fallen to 54 percent of the standard, and it has remained at about that level since. (Page 27)
- In 2000, about 1 million children experienced an average PM-10 concentration above the annual standard, down from about 2 million in 1990. (Page 28)
- In 1996, all children lived in counties in which the combined estimated concentrations of hazardous air pollutants exceeded the 1-in-100,000 cancer risk benchmark. Approximately 95 percent of children lived in counties in which at least one hazardous air pollutant exceeded the benchmark for health effects other than cancer. (Page 31)

Indoor Air Pollutants

- The percentage of homes with children under 7 in which someone smokes on a regular basis decreased from 29 percent in 1994 to 19 percent in 1999. (Page 33)

Drinking Water Contaminants

- The percentage of children served by public water systems that reported exceeding a Maximum Contaminant Level or violated a treatment standard decreased from 20 percent in 1993 to 8 percent in 1999. Every category of violation decreased between 1993 and 1999 except for nitrates and nitrites, which remained steady. (Page 37)
- In 1993, approximately 22 percent of children lived in an area served by a public water system that had at least one major monitoring and reporting violation. This figure decreased to about 10 percent in 1999. The largest number of monitoring and reporting violations occurred for the lead and copper standards. (Page 39)

Pesticide Residues

- From 1994 to 2001, the percentage of food samples with detectable organophosphate pesticide residues ranged between 19 percent and 29 percent. The highest detection rates were observed during 1996 and 1997, while the lowest detection rate was observed in 2001. (Page 41)

Land Contaminants

- As of September 2000, about 0.8 percent of children lived within one mile of a Superfund site listed on the National Priorities List (NPL) that had not yet been cleaned up or controlled, down from about 1.3 percent in 1990. As of September 2000, about 1.3 percent of children lived within one mile of any Superfund site listed on the Superfund NPL. (Page 43)

Part 2: Body Burdens

Concentrations of Lead in Blood

- The median (50th percentile) concentration of lead in the blood of children 5 years old and under dropped from 15 micrograms per deciliter (µg/dL) in 1976-1980 to 2.2 µg/dL in 1999-2000, a decline of 85 percent. (Page 55)
- The concentration of lead in blood at the 90th percentile in children 5 years old and under, representing the most highly exposed 10 percent of children in that age group, dropped from 25 µg/dL in 1976-1980 to 4.8 µg/dL in 1999-2000. (Page 55)
- Concentrations of lead in children's blood differ by race/ethnicity and family income. In 1999-2000, the median blood lead level in children ages 1-5 was 2.2 µg/dL. The median blood lead level for children living in families with incomes below the poverty level was 2.8 µg/dL and for children living in families above the poverty level it was 1.9 µg/dL. For all income levels, Black non-Hispanic children had a median blood lead level of 2.8 µg/dL. White non-Hispanic children had a median blood lead level of 2.1 µg/dL and Hispanic children had a median blood level of 2.0 µg/dL. (Page 57)
- Approximately 300,000 children ages 1-5 (about 2 percent) had a blood lead level of 10 µg/dL or greater in 1999-2000. (Page 59)

Concentrations of Mercury in Blood

- EPA has determined that children born to women with blood concentrations above 5.8 parts per billion are at some increased risk of adverse health effects. About 8 percent of women of child-bearing age had at least 5.8 parts per billion of mercury in their blood in 1999-2000. (Page 61)

Concentrations of Cotinine in Blood

- Cotinine is a marker of exposure to environmental tobacco smoke. In 1999-2000, median (50th percentile) levels of cotinine measured in children were 56 percent lower than they were in 1988-1991. Cotinine values at the 90th percentile, representing the most highly exposed 10 percent of children, declined by 18 percent between 1988-91 and 1999-2000. (Page 63)

Key Findings

Part 3: Childhood Illnesses

Respiratory Diseases

- Between 1980 and 1995, the percentage of children with asthma doubled, rising from 3.6 percent in 1980 to 7.5 percent in 1995. A decrease in the percentage of children with asthma occurred between 1995 and 1996, but interpreting single-year changes is difficult. (Page 71)
- In 2001, 8.7 percent (6.3 million) of all children had asthma. (Page 71)
- The percentage of children with asthma differs by race/ethnicity and family income. In 1997-2000, more than 8 percent of Black non-Hispanic children living in families with incomes below the poverty level had an asthma attack in the previous 12 months. Approximately 6 percent of White non-Hispanic children and 5 percent of Hispanic children living in families with incomes below the poverty level had an asthma attack in the previous 12 months. (Page 73)
- More than 6 percent of children living in families with incomes below the poverty level had an asthma attack in the previous 12 months. About 5 percent of children living in families with incomes at the poverty level and higher had an asthma attack in the previous 12 months. (Page 73)
- Emergency room visits for asthma and other respiratory causes were 369 per 10,000 children in 1992 and 379 per 10,000 children in 1999. (Page 75)
- Hospital admissions for asthma and other respiratory causes were 55 per 10,000 children in 1980 and 66 per 10,000 children in 1999. (Page 77)

Childhood Cancer

- The frequency of new childhood cancer cases has been fairly stable since 1990. The age-adjusted annual incidence of cancer in children increased from 128 to 161 cases per million children between 1975 and 1998. Cancer mortality decreased from 51 to 28 deaths per million children during the 1975-1998 period. (Page 79)
- Leukemia was the most common cancer diagnosis for children from 1973-1998, representing about 20 percent of the total childhood cancer cases. Incidence of acute lymphoblastic leukemia was 24 cases per million in 1974-1978 and approximately 28 cases per million in 1994-1998. Incidence of acute myeloid leukemia was approximately 5 cases per million in 1974-98 and about the same in 1994-98. (Page 81)

Neurodevelopmental Disorders

- In 1997-2000, about 6 children out of every 1,000 (0.6 percent) were reported to have been diagnosed with mental retardation. (Page 87)

Part 4: Emerging Issues

Mercury in Fish

- Since 1995, most states have issued one or more advisories to warn people about elevated concentrations of mercury in non-commercial fish. In some cases, advisories tell people to avoid eating fish that they catch in particular areas or to avoid particular species. In other cases, they tell people to limit the amount of fish that they consume. Some advisories are directed at particularly susceptible groups, usually women of child-bearing age and children. (Page 96)

Attention-Deficit/Hyperactivity Disorder

- In 1997-2000, 6.7 percent of children ages 5-17 were reported to have been diagnosed with attention-deficit/hyperactivity disorder (ADHD). (Page 99)

Part 5: Special Features

Lead in California Schools

- Thirty-two percent of all public elementary schools surveyed in California had both lead-based paint and some deterioration of paint. (Page 107)
- Eighty-nine percent of all California schools studied had detectable levels of lead in soils. Only 7 percent of the schools had lead levels in soil at or exceeding the EPA hazard standard. (Page 109)
- Approximately 15 percent of schools had lead levels in drinking water that exceeded EPA's drinking water standard on the first draw. Drinking water from approximately 6.5 percent of schools remained above the standard on the second draw. Second draw samples are more representative of the lead concentrations that children are exposed to during most of the day. (Page 111)

Pesticides in Minnesota Schools

- Approximately 47 percent of responding school custodians in Minnesota reported that they sprayed pesticides "as needed" in the classroom. Forty percent of the responding custodians reported that their schools provided no notification of pesticide use (such as notices in fumigated areas or pre- and postapplication letters to students and teachers). (Page 115)

Birth Defects in California

- Heart defects are the most common birth defect in California, with 1.8 cases per 1,000 live births in 1997-99. The rates of birth defects in California generally remained constant during the 1990s. (Page 117)

Summary List of Measures

Summary List of Measures Included in this Report

Name	Description of Measure	Year(s)
Environmental Contaminants		
Outdoor Air Pollutants		
Common Air Pollutants	Percentage of children living in counties in which air quality standards were exceeded	1990-2001
	Percentage of children's days with good, moderate, or unhealthy air quality	1990-1999
	Long-term trends in annual average concentrations of criteria pollutants	1990-2000
	Number of children living in counties with high annual averages of PM-10	1990-2000
Hazardous Air Pollutants	Percentage of children living in counties where estimated hazardous air pollutant concentrations were greater than health benchmarks	1996
Indoor Air Pollutants		
Environmental Tobacco Smoke	Percentage of homes with children under 7 where someone smokes regularly	1994-1999
Drinking Water Contaminants		
Drinking Water Contaminants	Percentage of children living in areas served by public water systems that exceeded a drinking water standard or violated treatment requirements	1993-1999
Monitoring and Reporting	Percentage of children living in areas with major violations of drinking water monitoring and reporting requirements	1993-1999
Pesticide Residues	Percentage of fruits, vegetables, and grains with detectable residues of organophosphate pesticides	1994-2001
Land Contaminants	Percentage of children residing within one mile of a Superfund site	1990-2000
Body Burdens		
Concentrations of Lead in Blood	Concentration of lead in blood of children ages 5 and under	1976-2000
	Median concentrations of lead in blood of children ages 1-5, by race/ethnicity and family income	1999-2000
	Distribution of concentrations of lead in blood of children ages 1-5	1999-2000
Concentrations of Mercury in Blood	Distribution of concentrations of mercury in blood of women of child-bearing age	1999-2000
Concentrations of Cotinine in Blood	Concentrations of cotinine in blood of children	1988-2000

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Summary List of Measures

Name	Description of Measure	Year(s)
Childhood Illnesses		
Respiratory Diseases	Percentage of children with asthma	1980-2001
	Percentage of children having an asthma attack in the previous 12 months, by race/ethnicity and family income	1997-2000
	Children's emergency room visits for asthma and other respiratory causes	1992-1999
	Children's hospital admissions for asthma and other respiratory causes	1980-1999
Childhood Cancer	Cancer incidence and mortality for children under 20	1975-1998
	Cancer incidence for children under 20 by type	1974-1998
Neurodevelopmental Disorders	Children reported to have mental retardation, by race/ethnicity and family income	1997-2000
Special Features		
Lead in California Schools	Percentage of California public elementary schools with lead paint and some deterioration of paint	1994-1997
	Percentage of California public elementary schools with lead in soils	1994-1997
	Percentage of California public elementary schools with lead in drinking water	1994-1997
Pesticide Use in Minnesota Schools	Frequency of application of pesticides in Minnesota K-12 schools	1999
Birth Defects in California	Number of birth defects in California per 1,000 live births and fetal deaths	1991-1999

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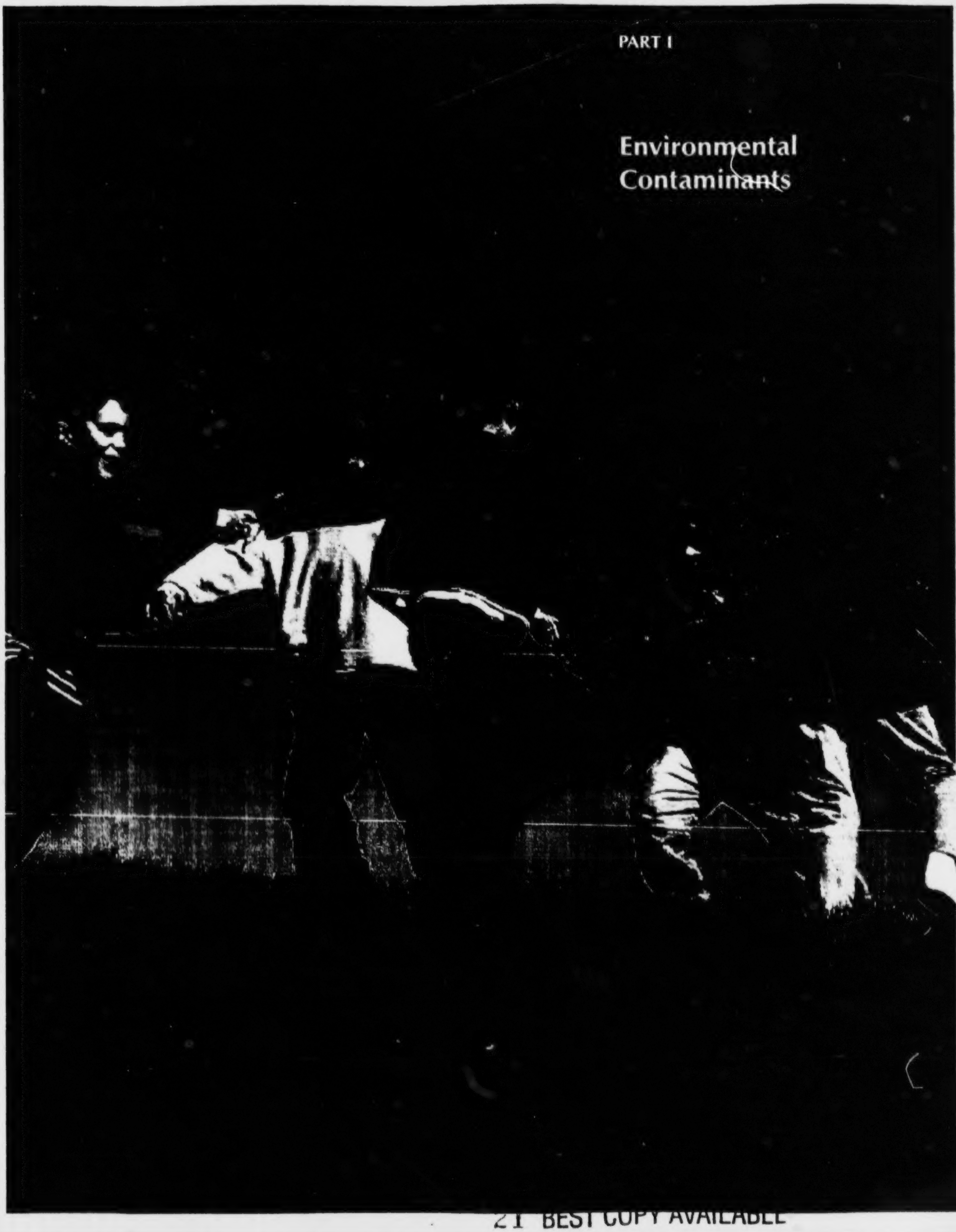
PART I

Environmental Contaminants



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Environmental Contaminants

Tracking environmental contaminants is an important step toward determining whether environmental policies protect children. This section of the report presents information about environmental contaminants that can affect children and discusses how levels of these contaminants in the environment have changed over time.

Pollutants or contaminants that can affect the health of children can be found in air, water, food, and soil. This section includes measures for contaminants in these media. Most of the measures show the percentages of children who may be at risk from exposure to critical concentrations of pollutants.

This second edition of *America's Children and the Environment* includes several new measures that reflect pollutants in environmental media.

The report adds a new measure that describes trends in long-term concentrations of pollutants in the air. This measure builds on the report's first two measures for air pollution, which reflect daily exposures to air pollutants. Research suggests that exposure to a few days of high concentrations of air pollutants or to many days of lower concentrations both can have adverse effects on health. The report also includes a new measure concerning pesticide residues in foods.

Describing the significance of pollutants in soils is a difficult problem because contamination often is localized and difficult to capture in a national report. To improve coverage of contaminants in soil, this report replaces an earlier measure that showed the percentages of children living in counties with a Superfund site with a new measure showing the percentage of children who live within a mile of a Superfund site.

This report does not assess quantitative relationships between the measures for environmental contaminants and childhood illnesses. The report includes a qualitative discussion of the research that has looked at some of these relationships.

The measures in this section do not account for many environmental contaminants that are important for children but lack nationally representative data. Such contaminants include those in dusts and soils in and near homes, and contaminants in soil from sources other than Superfund sites. The measure on food contaminants addresses only a few of the contaminants found in foods: selected pesticides used on certain items of produce. The measure does not account for pathways, other than the diet, by which children are exposed to pesticides. For example, pesticides may be transported into homes from outdoors or from the workplace on skin, clothing, or shoes. Children then may ingest pesticides when they put their hands in their mouths after touching contaminated surfaces or when they put objects in their mouths that have been contaminated with pesticides.

The data used to develop the measures within this section vary in coverage and completeness, as summarized in the chart on the next page.

Coverage of Environmental Contaminant Measures

Topic	Description of Measure	Year(s)	Geographic Coverage	Notes
Outdoor Air Pollutants				
Common Air Pollutants	E1: Percentage of children living in counties in which air quality standards were exceeded	1990-2001	County-level data	Measure includes five common (criteria) air pollutants. Many counties monitored only some common air pollutants and some counties did not monitor any.
	E2: Percentage of children's days with good, moderate, or unhealthy air quality	1990-1999	County-level data	Measure includes five common air pollutants.
	E3: Long-term trends in annual average concentrations of criteria pollutants	1999-2000	County-level data	Measure includes three common air pollutants.
Hazardous Air Pollutants	E4: Percentage of children living in counties where estimated hazardous air pollutant concentrations were greater than health benchmarks in 1996	1996	County-level data	Data for one year only; measure is based on estimates of ambient concentrations of 33 of the 188 hazardous air pollutants identified in the Clean Air Act.
Indoor Air Pollutants				
Environmental Tobacco Smoke	E5: Percentage of homes with children under 7 where someone smokes regularly	1994-1999	National-level data	Measure is a surrogate for environmental tobacco smoke in the home. Other indoor pollutants (e.g., combustion products, volatile organic compounds) would be relevant to include if data could be identified.
Drinking Water Contaminants				
Drinking Water Contaminants	E6: Percentage of children living in areas served by public water systems that exceeded a drinking water standard or violated treatment requirements	1993-1999	County-level data	Data on violations of standards are incomplete due to monitoring and reporting limitations. Measure is a surrogate for concentrations of contaminants.
Monitoring and Reporting	E7: Percentage of children living in areas with major violations of drinking water monitoring and reporting requirements	1993-1999	County-level data	Measure shows percentage of children living in areas where no information on drinking water contaminants is available; children may or may not be at risk.
Food Contaminants				
Pesticide Residues	E8: Percentage of fruits, vegetables, and grains with detectable residues of organophosphate pesticides	1994-2001	National-level data	Surrogate for dietary exposure to residues of organophosphate pesticides. Other contaminants in food, such as other pesticides and industrial chemicals that are relevant to children, are not included.
Land Contaminants				
Hazardous Waste Sites	E9: Percentage of children residing within one mile of a Superfund site	1990-2000	Site-specific locations	Does not reflect exposures from sites that may be hazardous but are not included on the Superfund National Priorities List. Proximity to a Superfund site does not necessarily indicate that children are exposed to contaminants.

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Common ("Criteria") Air Pollutants

Air pollution contributes to a wide variety of adverse health effects. Six of the most common air pollutants—carbon monoxide, lead, ground-level ozone, particulate matter, nitrogen dioxide, and sulfur dioxide—are known as "criteria" pollutants because EPA uses health-based criteria as the basis for setting permissible levels of these pollutants in the atmosphere.

EPA periodically conducts comprehensive reviews of the scientific literature on health effects associated with exposure to the criteria air pollutants. The resulting "criteria documents" critically assess the scientific literature and serve as the basis for making regulatory decisions about whether to retain or revise the National Ambient Air Quality Standards (NAAQS) that specify the allowable concentrations of each of these pollutants in the air. The standards are set at a level that protects public health with an adequate margin of safety. However, the standards are not "risk free." Even in areas that meet the standards, there may be days when unusually sensitive individuals, including children, experience health effects related to air pollution. This is especially the case for pollutants such as ozone and particulate matter that do not have discernible thresholds below which health effects are absent.

Some of the standards are designed to protect the public from adverse health effects that can occur after being exposed for a short time, such as one hour or one day. Other standards are designed to protect people from health effects that can occur after being exposed for a much longer time, such as a year. For example, current standards for carbon monoxide are for short-term periods of one hour and eight hours. By contrast, the current standard for nitrogen dioxide is for one year. The standards and the varying time periods for which they apply are shown in Table 1 in Appendix B. Some pollutants have both short-term and long-term standards.

Health effects that have been associated with each of these pollutants are summarized below. This information is drawn from EPA's criteria documents as well as more recent studies.

Ground-level Ozone

Short-term (also known as "acute") exposure to ground-level ozone can cause a variety of respiratory health effects, including inflammation of the lung, reduced lung function, and respiratory symptoms such as cough, chest pain, and shortness of breath. It also can decrease the capacity to perform exercise.[1] Exposure to ambient concentrations of ozone also has been associated with the exacerbation of asthma, bronchitis, and respiratory effects serious enough to require emergency room visits and hospital admissions.[1] Some evidence suggests that high ozone concentrations may contribute to increased mortality.[1]

Health effects associated with long-term (also known as "chronic") exposure to ozone are not as well established and documented as health effects associated with short-term exposure, but long-term exposures also are of concern. In 1996, EPA's criteria document for ozone concluded that there was insufficient evidence to determine whether health effects resulted directly from long-term exposure, although the evidence suggested that long-term ozone exposure, along with other environmental factors, could be responsible for health effects.[1] Since 1996, a few studies suggest that long-term exposure to ozone is associated with decreases in lung function in humans,[2] increased prevalence of asthma,[3] increased development of asthma in children who exercise outdoors,[4] and exacerbation of existing asthma.[5]

Outdoor Air Pollutants

Particulate Matter

Particulate matter in the air (often called PM-10 or PM-2.5) has been found to cause increased risk of mortality (death), hospital admissions and emergency room visits for heart and lung diseases, respiratory effects, and decreases in lung function.[6] Such health effects have been associated with both short-term and long-term exposure to particulate matter. Children and adults with asthma are considered to be among the groups most sensitive to respiratory effects.[6-10] Studies published since the release of EPA's criteria document for particulate matter have found further evidence of an association between particulate matter and increased respiratory disease and symptoms in children with asthma[11] and increased hospitalizations or emergency room visits for persons with asthma.[5, 12, 13] Studies also have confirmed that chronic exposure to particulate matter is associated with mortality in adults[14-16] and suggest that it may be associated with mortality in infants.[17] Also, recent studies suggest that chronic exposure to particulate matter may affect lung function and growth.[18, 19]

Prior to 1997, the National Ambient Air Quality Standard for particulate matter was based on particulate matter measuring 10 microns or less (PM-10). In 1997, the standard was revised to address the health risks from particulate matter measuring 2.5 microns or less (PM-2.5).

Lead

Lead accumulates in bones, blood, and soft tissues of the body. Exposure to lead can affect development of the central nervous system in young children, resulting in neurobehavioral effects such as lowered IQ.[20]

Sulfur Dioxide

Sulfur dioxide poses particular concerns for those with asthma, who are considered to be especially susceptible to its effects.[21] Short-term exposures of asthmatic individuals to elevated levels of sulfur dioxide while exercising at a moderate level may result in breathing difficulties accompanied by symptoms such as wheezing, chest tightness, or shortness of breath. Effects that have been associated with longer-term exposures to high concentrations of sulfur dioxide, in conjunction with high levels of particulate matter include respiratory illness, alterations in the lung's defenses, and aggravation of existing cardiovascular diseases.

Carbon Monoxide

Exposure to carbon monoxide reduces the capacity of the blood to carry oxygen, thereby decreasing the supply of oxygen to tissues and organs such as the heart. Short-term exposure can cause effects such as reduced time to onset of angina pain, neurobehavioral effects, and a reduction in exercise performance.[22] Long-term exposure has not been studied adequately in humans to draw conclusions regarding possible chronic effects, though a recent study reported an association between long-term exposure to carbon monoxide and other traffic-related pollutants and respiratory symptoms in children.[23]

Nitrogen Dioxide

Exposure to nitrogen dioxide has been associated with a variety of health effects.[24] Effects include decreased lung function,[23, 25, 26] increased respiratory symptoms or illness,[7, 23, 27-29] and increased symptoms in children with asthma.[11] Nitrogen dioxide also is a major contributor to the formation of ground-level ozone.[1]

Exceedances of Short-Term Air Quality Standards

State agencies that monitor air quality report their findings to EPA. In turn, EPA compares the measured values reported by states to the National Ambient Air Quality Standards in order to determine whether pollutants exceed the established standards. EPA uses the term "exceedance" to refer to a case in which a reported measurement of a pollutant is higher than the standard. Appendix B includes a description of the methods used to determine whether an exceedance has occurred.

This measure uses EPA data on exceedances of short-term air quality standards in counties in the United States. This data source simply indicates whether each standard was exceeded at any time during a year. This measure shows the percentage of children living in areas with any such exceedances, who thus may be exposed to poor daily air quality at some point during a year. In addition, the measure includes exceedances of the new ozone and particulate matter standards adopted in 1997. The ozone standard is based on an eight-hour average ozone value. The new particulate matter standards are for PM-2.5 and have both annual and 24-hour averaging periods. The annual PM-2.5 standard is intended to protect against both short-term and long-term health effects.

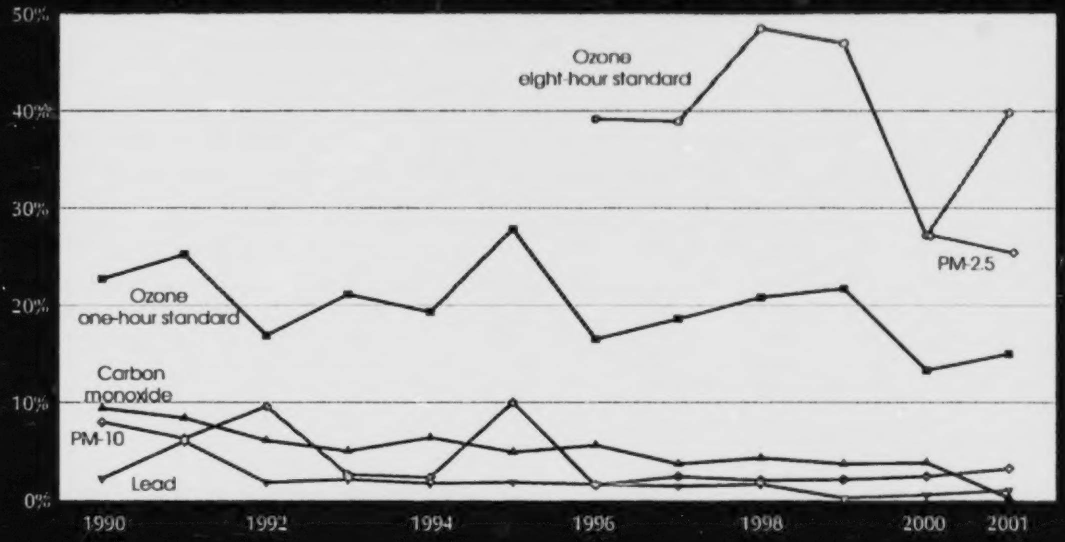
This measure does not differentiate between areas in which standards are exceeded frequently or by a large margin, and areas in which standards are exceeded only rarely or by a small margin. The measure is based on exceedances of individual standards and does not reflect any combined effect of multiple pollutants. Also, because the nature of health effects varies significantly and the averaging times associated with different standards vary widely, exceedances for different standards are not comparable. For example, the ozone standard considers measured levels of ozone within a one-hour or eight-hour period and health effects such as lung function decrements, respiratory symptoms, and hospital admissions. In contrast, the averaging time for the lead standard is three months and is based on health effects such as IQ decrements and hypertension.

The graph shows the percentage of children who live in counties with exceedances for any of four of the six criteria pollutants. Nitrogen dioxide is not included, as there is no short-term standard for this compound. Sulfur dioxide also is not shown, since few exceedances have been reported since 1993.

Healthy People 2010: Objective 8-01 of Healthy People 2010 aims to reduce the proportion of persons exposed to air that exceeds the levels of U.S. Environmental Protection Agency's health-based standards for harmful air pollutants. See Appendix C for more information.

Measure E1

Percentage of children living in counties in which air quality standards were exceeded



SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

- The highest number of exceedances is consistently reported for ozone. In 1990, approximately 23 percent of children lived in counties in which the one-hour ozone standard was exceeded on at least one day per year. In 2001, approximately 15 percent of children lived in such counties. Exceedances of the eight-hour ozone standard are reported beginning in 1996. In 1996-2001, significantly more children lived in counties that exceeded the eight-hour ozone standard than in counties that exceeded the one-hour standard.
- In 2000, approximately 27 percent of children lived in counties that exceeded the annual PM-2.5 standard. In 2001, approximately 25 percent of children lived in such counties. (The standard is intended to protect against both short-term and long-term health effects and thus PM-2.5 is included in Measure E1.)
- In 1990, approximately 10 percent of children lived in counties in which the carbon monoxide standard was exceeded. In 2001, approximately 0.2 percent of children lived in such counties.
- From 1990 to 2001, the percentage of children living in counties that exceeded the one-day standard for PM-10 fluctuated, but was as high as 10 percent in 1992 and 1995. The percentage remained around 2 to 3 percent from 1996-2001.
- Since 1992, on average, 2 percent of children lived in counties that exceeded the three-month standard for lead. In 2000, the three counties with reported lead exceedances were Madison County, Illinois; Jefferson County, Missouri; and Dallas County, Texas.
- Few exceedances of the sulfur dioxide standard have occurred since 1993. Consequently, it was not included on the graph.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants (E1-E3)		Respiratory (D1-D4)	
	Criteria Air Pollutants: lead (E1-E2) *Drinking Water: lead (E6-E7)*	Lead in Blood (B1-B3)	Neurodevelopmental (D7)	Lead in Schools (S1-S3)

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Daily Air Quality

EPA provides an Air Quality Index (AQI) that represents air quality for specific days and is widely reported in newspapers and other media outlets in metropolitan areas.

The AQI is based on measurements of up to five of the six air quality criteria pollutants (carbon monoxide, ground-level ozone, nitrogen dioxide, particulate matter, and sulfur dioxide). Lead is not included in the AQI. The specific pollutants considered in the AQI for each metropolitan area depend on which pollutants are monitored in that area. Each pollutant concentration is given a value on a scale that is related to the air quality standards for that pollutant. An AQI value of 100 for a criteria pollutant generally corresponds to the short-term National Ambient Air Quality Standard for that pollutant, and is the level EPA has set to protect public health for a single day. Above this level, pollutant-specific health advisories are issued. The daily AQI is based on the pollutant with the highest index value on the scale that day. It does not add up values for more than one pollutant. Therefore, it does not reflect the possible effects of simultaneous exposure to high levels of multiple pollutants.

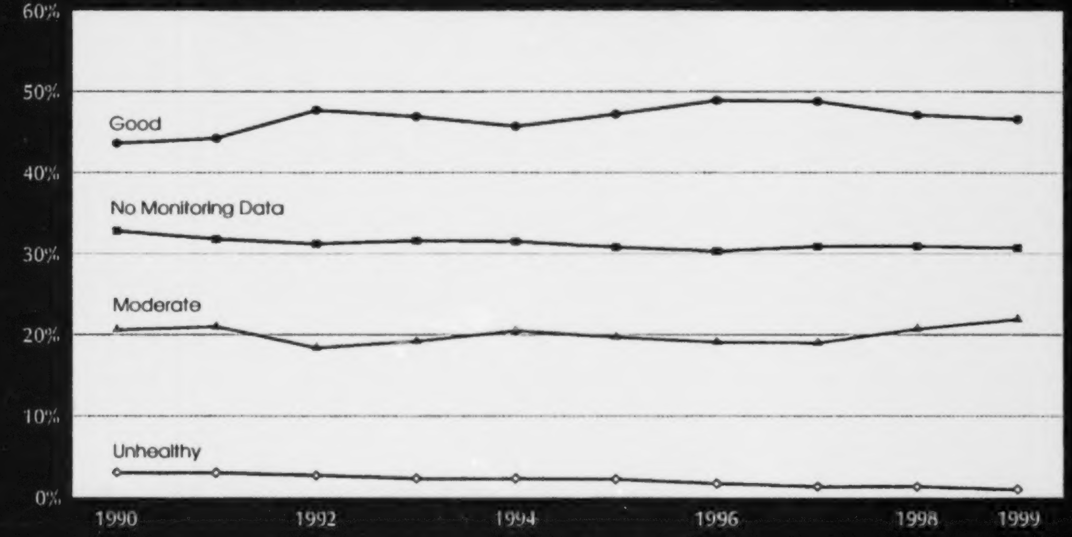
EPA has divided the AQI scale into categories. Air quality is considered "good" if the AQI is between 0 and 50, posing little or no risk. Air quality is considered "moderate" if the AQI is between 51 and 100. Some pollutants at this level may present a moderate health concern for a small number of individuals. Moreover, such a level may pose health risks if maintained over many days. Air quality is considered "unhealthy for sensitive groups" if the AQI is between 101 and 150. Members of sensitive groups such as children may experience health effects, but the general population is unlikely to be affected. Air quality is considered "unhealthy" if the AQI is between 151 and 200. The general population may begin to experience health effects, and members of sensitive groups may experience more serious health effects.

Measure E2 on the following page is based on the reported AQI for counties of the United States. (Not all counties have air quality monitoring stations.) This measure was developed by reviewing the air quality designation for each day for each county and weighting the daily designations by the number of children living in each county. The overall measure reports the percentage of children's days of exposure considered to be of good, moderate, or unhealthy air quality.

The advantage of this approach, compared with that used in measure E1, is that it provides a sense of the intensity of pollution over the course of a year. This method provides data on the air quality category for each day, rather than simply reporting whether a county ever exceeds any standard for any pollutant. However, the method has some limitations. The AQI is based on the single pollutant with the highest value for each day; it does not reflect any combined effect of multiple pollutants. It reflects only short-term, daily pollution burdens. It does not include lead. The approach is influenced by the frequency of measurements. Because the AQI is reported daily, pollutants that are measured daily—such as ozone—will appear to have more effect than those that are measured less frequently, such as PM-10, which typically is measured every six days. Also, the AQI is not well-suited for reporting concentrations of nitrogen dioxide, because this pollutant does not have a short-term standard.

Measure E2

Percentage of children's days with good, moderate, or unhealthy air quality



SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

- The percentage of days that were designated as having "unhealthy" air quality (including days that were unhealthy for everyone as well as those that were unhealthy for sensitive groups) decreased between 1990 and 1999, dropping from 3 percent in 1990 to less than 1 percent in 1999. The percentage of days with "moderate" air quality remained around 20 percent between 1990 and 1999, although an upward trend is suggested by the fact that the percentage of moderate air quality days was higher in 1999 than for any other year in this analysis. As the percentage of either unhealthy or good air days decreases, the percentage of moderate days would be expected to increase.
- The coverage of monitoring for this measure, in terms of area and percentage of days monitored, was largely unchanged between 1990 and 1999. Approximately 30 percent of children's days of exposure to air pollutants were not monitored. This percentage includes days for which no AQI was reported in counties where the AQI is sometimes reported, as well as counties in which the AQI is not reported at all. On days that were monitored, in many cases only one or a few pollutants were monitored.

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Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants (E1-E3)		Respiratory (D1-D4)	

Long-Term Exposure to Criteria Air Pollutants

Most measures used to describe air pollution focus on days when pollutant levels are high. This approach is appropriate because high pollution levels over short periods of time, even less than a day, can contribute to many adverse health effects.[1]

Accumulated exposures to criteria air pollutants over longer periods of time also may affect health. As it has for short-term exposures, EPA has set standards for longer time periods for some pollutants. The concentrations of air pollutants in the long-term air pollution standards established by EPA are not "risk-free." Even in areas that meet the standards, unusually sensitive individuals, including children, may experience health effects related to air pollution. This is especially true for pollutants, such as particulate matter, that do not have discernible thresholds below which health effects are absent. Comparisons of pollutant concentrations with longer-term air quality standards can help identify the pollutants that pose the greatest concerns. Such comparisons can provide a perspective on whether pollutants pose equal or different levels of concern with regard to long-term exposure.

Measure E3a presents trends in the long-term exposures of children to three of the six criteria pollutants that have long-term standards: particulate matter, sulfur dioxide, and nitrogen dioxide. This measure reflects annual averages of pollutants. It shows how the average exposure of children compares with the applicable long-term National Ambient Air Quality Standard, and how the long-term exposure has changed over the last several years.

The values shown in Measure E3a are all based on standards for individual pollutants, and do not reflect any combined effect of multiple pollutants. For nitrogen dioxide and sulfur dioxide, data are available only for 1996-1999. For PM-10, data are available for 1990-2000. EPA adopted a new standard for PM-2.5 (finer particles) in 1997, but several years of data on PM-2.5 were not available for this report.

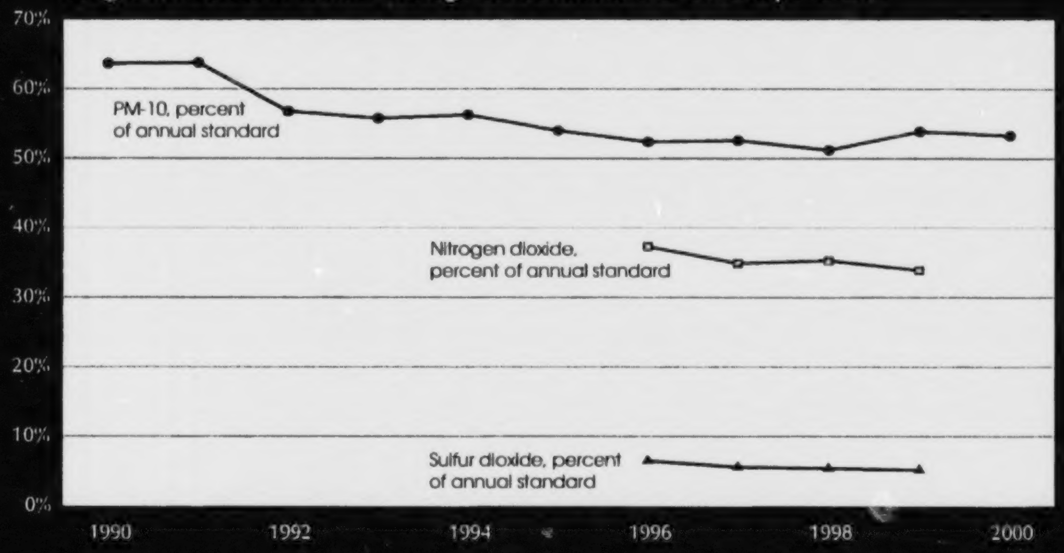
Measure E3b shows the number of children living in counties where long-term standards for PM-10 have been exceeded. Exposure to particulate matter has increasingly been recognized as a health concern. Health effects have been documented at concentrations that are experienced in the United States today.

In addition, as noted above, research suggests that there is no "safe" level of particulate matter and that any exposure poses some risk.[6] It is therefore valuable to track the number of children who live in counties where particulate matter concentrations, while not exceeding the standard, are relatively high. Measure E3b provides additional perspective on the concentrations of PM-10 to which children are exposed, by showing the number of children who live in counties that exceed 80 percent of the long-term standard. This is a somewhat arbitrary cutoff, as a different value (such as 90 percent or 75 percent) could have been chosen. However, the measure does provide an indication of the percentage of children who are living in areas where measured concentrations are fairly close to the standard and are of interest for tracking purposes.

Outdoor Air Pollutants

Measure E3a

Long-term trends in annual average concentrations of criteria pollutants



SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

- In 1990, on average, children experienced a concentration of 31.9 μg/m³ of PM-10, which represents 64 percent of the standard for the year. By 1995, the concentration had fallen to 54 percent of the standard, and it has remained at about that level since. From 1990-2000, between 55 and 66 percent of children lived in counties with monitoring stations for PM-10.
- In 1996, on average, children experienced a concentration of 0.02 parts per million of nitrogen dioxide, which represents 37 percent of the standard for the year. By 1999, this percentage had fallen to 34 percent of the standard on average. During these years, between 45 and 47 percent of children lived in counties with monitoring stations for nitrogen dioxide.
- In 1996, on average, children living in counties with monitoring stations experienced a concentration of 0.002 parts per million of sulfur dioxide, which represents 6.5 percent of the standard for the year. By 1999, this percentage had fallen to 5.2 percent of the standard on average. During these years, between 31 and 36 percent of children lived in counties with monitoring stations for sulfur dioxide.

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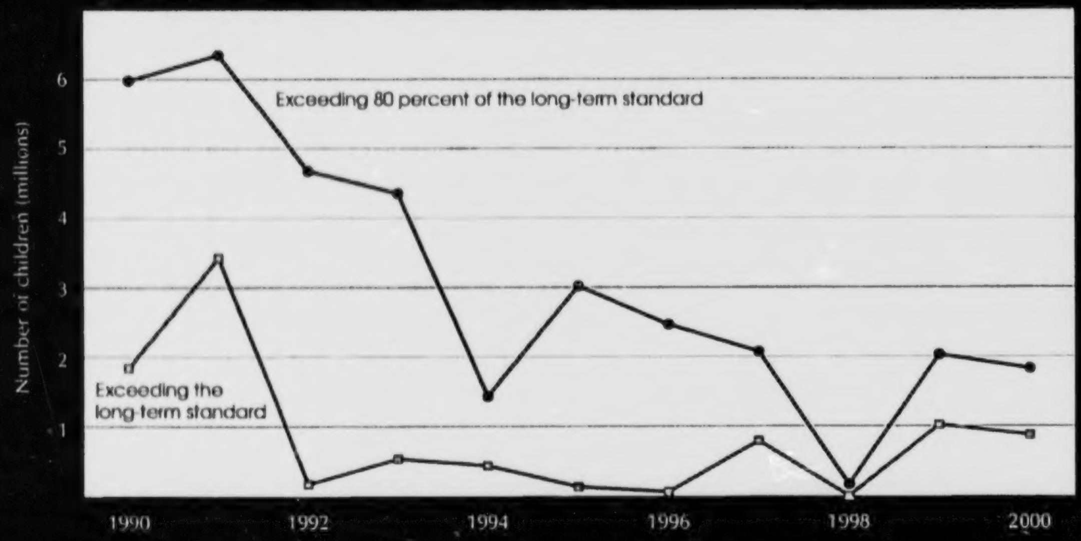
Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants (E1-E3)		Respiratory (D1-D4)	

Measure E3b

Number of children living in counties with high annual averages of PM-10



SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

- In 2000, about 1 million children experienced an average PM-10 concentration above the annual standard, down from about 2 million in 1990.
- In 2000, about 2 million children experienced a relatively high average concentration of PM-10 (greater than 80 percent of the annual standard), down from about 6 million children in 1990.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants (E1-E3)		Respiratory (D1-D4)	

Hazardous Air Pollutants

Hazardous air pollutants, also known as air toxics, have been associated with a number of adverse human health effects, including cancers, asthma and other respiratory ailments, and neurological problems such as learning disabilities and hyperactivity.[30-38]

The Clean Air Act identifies 188 substances as hazardous air pollutants. Examples include benzene, trichloroethylene, mercury, chromium, and dioxin. EPA establishes standards for hazardous air pollutant emissions for separate source categories. Hazardous pollutants are emitted from sources that are grouped into three general categories: major sources, area sources, and mobile sources. Major sources typically are large industrial facilities such as chemical manufacturing plants, refineries, and waste incinerators. These sources may release air toxics from equipment leaks, when materials are transferred from one location to another, or during discharge through emission stacks or vents. Area sources typically are smaller stationary facilities such as dry cleaners. Though emissions from individual area sources often are relatively small, collectively their emissions can be of concern—particularly where large numbers of sources are located in heavily populated areas. Mobile sources include both on-road sources, such as cars, light trucks, large trucks and buses, and non-road sources such as farm and construction equipment, lawn and garden equipment, marine engines, aircraft, and locomotives.

Unlike the criteria air pollutants, hazardous air pollutants have no national air quality standards that can be used to construct a health-based measure. Instead, the measure shown here compares estimates of ambient concentrations for 33 hazardous air pollutants with health benchmark concentrations derived from scientific assessments conducted by EPA and other environmental agencies.[32, 38-40]

Hazardous Air Pollutants and Health Benchmarks

This analysis compares ambient concentrations of hazardous air pollutants with three health benchmark concentrations. Two benchmarks reflect potential cancer risks, at levels of 1-in-100,000 risk and 1-in-10,000 risk. If a particular hazardous air pollutant is present in ambient air at a 1-in-100,000 benchmark concentration, for example, one additional case of cancer would be expected in a population of 100,000 people exposed for a lifetime. The third benchmark concentration corresponds to the level at which exposure to the hazardous air pollutant is judged to be of minimal risk; exposures above this benchmark may be associated with adverse health effects other than cancer.

The three benchmarks generally reflect health risks to adults, rather than potential risks to children or risks in adulthood stemming from childhood exposure. Benchmarks are not available to reflect the latter concerns. Further, the benchmarks reflect risks of continuous exposure over the course of a lifetime. Potential risks from very high short-term exposures, or from elevated exposures that may be experienced during childhood, are not addressed by these benchmarks. Therefore, this analysis does not represent a prediction of actual cancer rates in children.

The estimates of ambient concentrations of 33 air toxics for the year 1996 were generated as part of EPA's National Air Toxics Assessment. A computer model provided estimates for every county in the continental United States. The computer estimates generally are consistent with the limited set of actual measurements of ambient air toxics concentrations available for 1996, though at many locations the model estimates are lower than the measured concentrations.

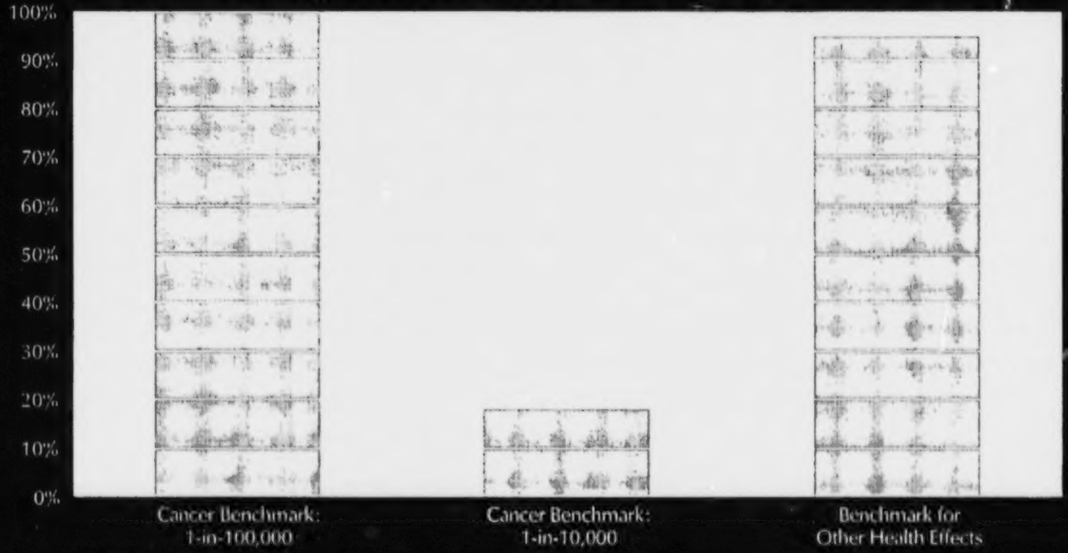
Actual exposures may differ from ambient concentrations. Indoor concentrations of hazardous air pollutants from outdoor sources may be slightly lower than ambient concentrations, though they can be significantly higher if any indoor sources are present. Levels of some hazardous pollutants may be substantially higher inside cars and school buses, and those higher levels would increase the risks.

This measure only considers exposures to air toxics that occur by inhalation. For many air toxics, dietary exposures also are important. Air toxics that are persistent in the environment settle out of the atmosphere onto land and water, and then accumulate in fish and other animals in the food web. For hazardous air pollutants that are persistent in the environment, exposures through food consumption typically are greater than inhalation exposures. Hazardous air pollutants for which these food chain exposures are important include mercury, dioxins, and PCBs.[41-43]

Healthy People 2010: Objective 8-04 of Healthy People 2010 focuses on reducing emissions of hazardous air pollutants. See Appendix C for more information.

Measure E4

Percentage of children living in counties where estimated hazardous air pollutant concentrations were greater than health benchmarks in 1996



SOURCE: U.S. Environmental Protection Agency, National Air Toxics Assessment

- In 1996, all children lived in counties in which the combined estimated concentrations of hazardous air pollutant exceeded the 1-in-100,000 cancer risk benchmark.
- Eighteen percent of children lived in counties in which hazardous air pollutants combined to exceed the 1-in-10,000 cancer risk benchmark. The pollutants that contributed most to this result were formaldehyde (mostly from mobile sources) and chromium (mostly from chromium electroplating). Formaldehyde is considered by EPA to be a "probable human carcinogen" and chromium is a "known human carcinogen."
- Approximately 95 percent of children lived in counties in which at least one hazardous air pollutant exceeded the benchmark for health effects other than cancer. In almost all cases, this result was attributable to the pollutant acrolein, which is a respiratory irritant. More than 75 percent of acrolein emissions are from mobile sources such as cars, trucks, buses, planes, and construction equipment.
- Exposures to diesel particulate matter are not included in this measure, because of uncertainty regarding the appropriate values to use as cancer benchmarks. Some studies have found that cancer risks from diesel particulate matter exceed those of the hazardous air pollutants considered in this measure.[44] Although EPA does not endorse any particular cancer benchmark value for diesel particulate matter, if the State of California's benchmark for diesel particulate matter were used in this analysis, 98 percent of children would live in counties where hazardous air pollutant estimates combined to exceed the 1-in-10,000 cancer risk benchmark.

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Environmental Tobacco Smoke: Smoking in the Home

Children can be exposed to a number of air pollutants that come from sources inside homes, schools, and other buildings. Indoor sources include combustion sources such as gas stoves, fireplaces, and cigarettes; building materials such as treated wood and paints, furnishings, carpet, and fabrics; and consumer products such as sprays, pesticides, window cleaners, and laundry soap. Indoor air pollutants also can come from outside, as air pollution penetrates indoors. Information on the toxic effects of air pollutants from indoor sources indicates that they could pose health risks to children.[45, 46]

Children who are exposed to environmental tobacco smoke, also known as secondhand smoke, are at increased risk for a number of adverse health effects, including lower respiratory tract infections, bronchitis, pneumonia, fluid in the middle ear, asthma symptoms, and sudden infant death syndrome (SIDS).[47-52] Exposure to environmental tobacco smoke also may be a risk factor contributing to the development of new cases of asthma.[48, 53, 54] Young children appear to be more susceptible to the effects of environmental tobacco smoke than older children are.[46, 48]

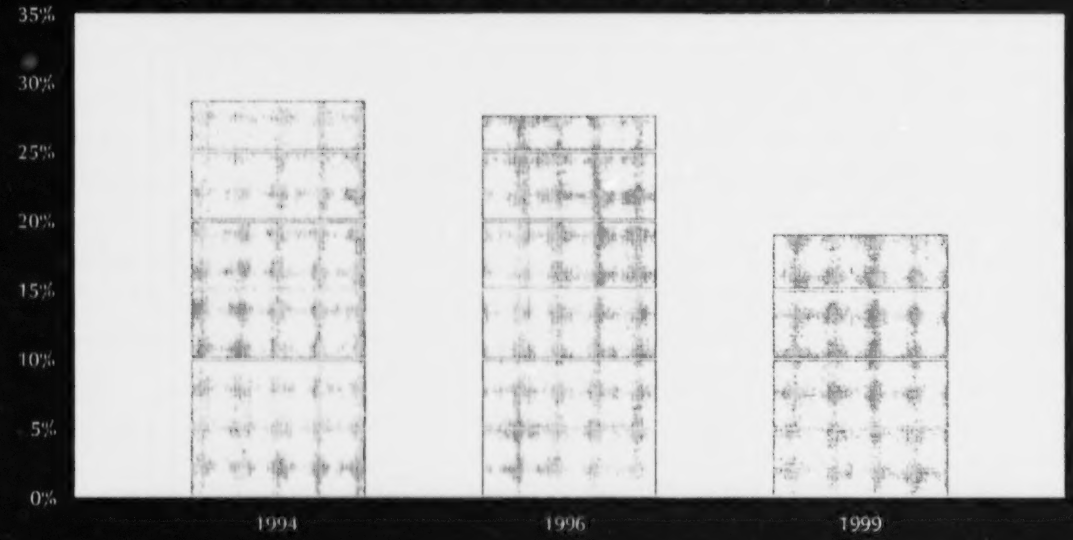
Smoking in the home is an important source of exposure because young children spend most of their time at home and indoors. The measure for environmental tobacco smoke shows the percentage of homes with children under 7 in which someone smokes regularly. Most often the smoker in the home is a parent.

This measure is a surrogate for the exposure of children to tobacco smoke. The data come from a national survey and are available for 1994, 1996, and 1999. The measure reflects the percentage of homes, rather than children, although it is expected that the two would track closely.

Healthy People 2010: Objective 27-9 of Healthy People 2010 is to reduce the proportion of children who are regularly exposed to tobacco smoke at home. See Appendix C for more information.

Measure E5

Percentage of homes with children under 7 where someone smokes regularly



SOURCE U.S. Environmental Protection Agency, Office of Air and Radiation, Indoor Environments Division, Surveys on Radon Awareness and Environmental Tobacco Issues

- The percentage of homes with children under 7 in which someone smokes on a regular basis decreased from 29 percent in 1994 to 19 percent in 1999.
- This measure shows the percentage of homes where a regular smoker lives. However, some percentage of these regular smokers may not actually smoke inside the home. The percentage of children who are exposed to secondhand smoke in the home thus may be smaller than suggested by the graph above.
- In 1999, an estimated 23.5 percent of adults were current smokers, down from 25.0 percent in 1993.[55]

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Environmental Tobacco Smoke (E5)	Cotinine in Blood (B5)	Respiratory (D1-D4)	

Contaminants in Children's Drinking Water

The contaminants in drinking water are quite varied and may cause a range of diseases in children, including acute diseases such as gastrointestinal illness, developmental effects such as learning disorders, and cancer.[56] Children are particularly sensitive to microbial contaminants because their immune systems are less developed than those of most adults.[56] Children are sensitive to lead, which affects brain development,[58-65] and to nitrates and nitrites, which can cause methemoglobinemia (blue baby syndrome).[66-68] Fertilizer, livestock manures, and human sewage are significant contributors of nitrates and nitrites in groundwater sources used for drinking water.[69-71]

EPA sets drinking water standards for public water systems, referred to as Maximum Contaminant Levels (MCLs).[72] These standards are designed to protect people against adverse health effects from contaminants in drinking water while taking into account the technical feasibility of meeting the standard and balancing costs and benefits. EPA has set MCLs for more than 80 microbial contaminants, chemicals, and radionuclides. EPA also has developed regulations to protect drinking water sources and to require treatment of drinking water. An important treatment-related regulation, the Surface Water Treatment Rule, requires treatment of surface waters used for drinking water by filtration to remove microbial contaminants.

Drinking water rules often are added or modified. For example, EPA established more stringent filter performance requirements in 1998 to further strengthen protection against microbial contaminants. In the same year, EPA also established new drinking water standards for disinfection byproducts, exposure to which has been associated with bladder cancer[73] and possible reproductive effects.[74] In 2000, EPA finalized standards protecting against radionuclides in drinking water.[75] In addition, EPA strengthened the existing standard for arsenic in 2001. Changes in regulatory requirements may affect the outcome of the measures presented in this report, as the resulting trends sometimes may be related to changes in standards rather than changes in exposures.

Exceedances of Drinking Water Standards

One way to measure children's risk of exposure to contaminated drinking water is to identify public water systems that contain contaminants at levels greater than those allowed by the drinking water standards. Ideally, concentrations for all chemical and microbial contaminants in all drinking water systems would be available for analysis to identify areas of risk for children. Currently this is not possible. The Safe Drinking Water Information System (SDWIS) does not track concentrations of contaminants in drinking water, but instead tracks the frequency with which standards are exceeded.

Public water systems are required to monitor individual contaminants at specific time intervals to assess whether they have achieved compliance with drinking water standards. When a violation of a drinking water standard is detected, the public water system is required to report the violation to state and federal governments. Information about exceedances can be used as a surrogate for exposure to unacceptably high levels of drinking water contaminants.

The reported violations received by the federal government are highly accurate, but violations may be under-reported in some cases because some public water systems fail to fully monitor contaminants or report their monitoring results. Data identifying public water systems that do not monitor or report their results are available. A review of the federal SDWIS database published in October 2000 found that 68 percent of the microbial contaminant violations, 19 percent of violations for other contaminants, and 11 percent of treatment and filtration violations that should be included in the SDWIS database are reported.[76] As a result of these findings, many states have taken corrective steps to improve their SDWIS data quality.

It also is important to consider information about water sources that are not included in the SDWIS database. Because data are only available for public water systems, this measure does not include children served by private water sources, such as wells or bottled water. Approximately 42 million people are served by private water systems that are not required to monitor and report the quality of drinking water.[77] Many people served by private water supplies live in rural and agricultural areas, which may be at increased risk for nitrate and nitrite contamination. Conversely, many children served by public water systems may not drink the tap water or may use a water filtration device to further purify the water. Thus, the measure may overestimate the percentage of children exposed to contaminated drinking water.

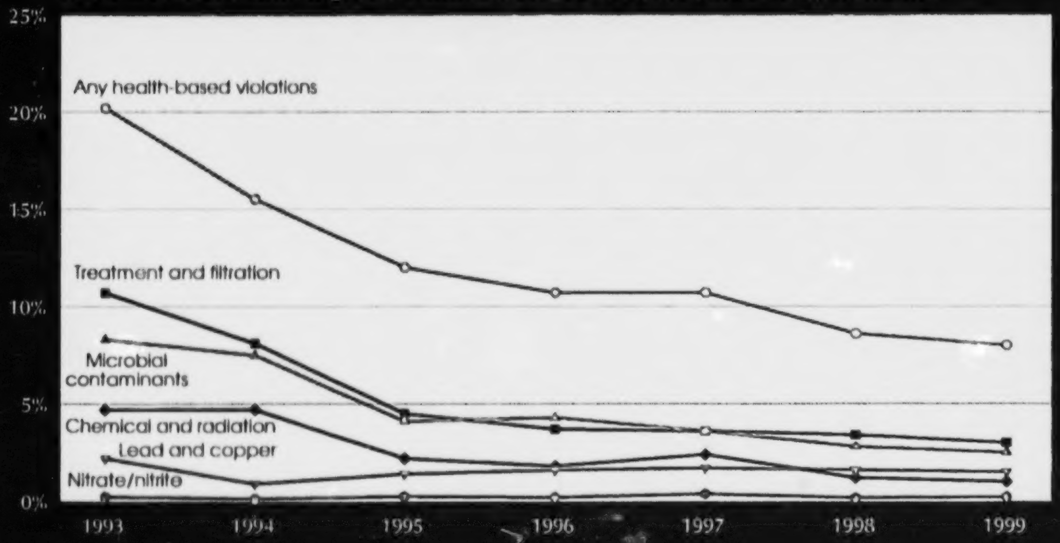
A violation of "treatment and filtration" is defined as any failure in the treatment process, or in operation and maintenance activities, or both, that may affect water quality.[78] The Surface Water Treatment Rule specifies the type of treatment and maintenance activities that systems must use to prevent microbial contamination of drinking water.

Healthy People 2010: Objective 8-05 of Healthy People 2010 seeks to increase the number of people served by community water systems that meet the regulations of the Safe Drinking Water Act. See Appendix C for more information.

Drinking Water Contaminants

Measure E6

Percentage of children living in areas served by public water systems that exceeded a drinking water standard or violated treatment requirements



SOURCE: U.S. Environmental Protection Agency, Office of Water, Safe Drinking Water Information System (Percentages are estimated)

- The percentage of children served by public water systems that reported exceeding a Maximum Contaminant Level or violated a treatment standard decreased from 20 percent in 1993 to 8 percent in 1999.
- Every category of reported violation decreased between 1993 and 1999 except for nitrates and nitrites, which remained steady. The largest decline was for violations of the treatment and filtration standards.
- From 1993-1999, approximately 0.2 percent of the children served by public water systems were served by systems that reported violations of the nitrate or nitrite standard.
- Between 1993 and 1999, fewer than 0.2 percent of all children served by public water systems were served by systems that had violations of the Total Trihalomethane (TTHM) standard. Four recent epidemiological studies have found significant associations between elevated TTHM exposure and stillbirth or miscarriages, but more study is necessary before any definitive conclusion can be made.[79-86]

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants: lead (E1-E2) *Drinking Water: lead (E6-E7)*	Lead in Blood (B1-B3)	Neurodevelopmental (D7)	Lead in Schools (S1-S3)

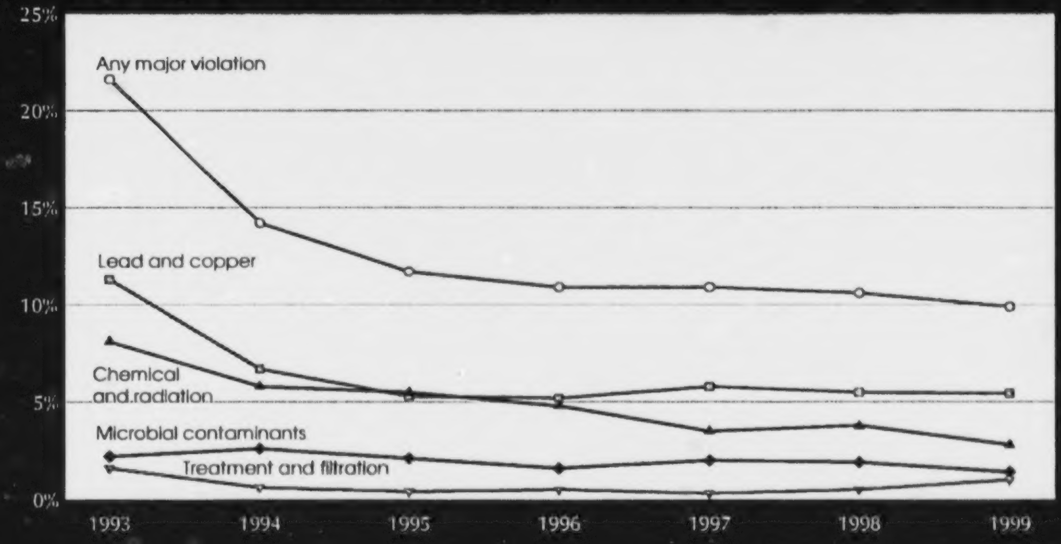
Monitoring and Reporting

Public water systems are required to monitor for contaminants and to report violations of drinking water standards to EPA. However, some public water systems do not conduct all of the required monitoring. Not all systems report violations. Such water systems violate monitoring and reporting requirements.

Some monitoring and reporting violations, such as late reporting, are minor. However, many water systems have major violations. For example, some water systems fail to collect any water samples during specified monitoring periods. Children who live in areas that are not adequately monitoring for water contaminants or reporting violations may be at risk, but the extent of any possible exposures in violations of drinking water standards and their associated risks is unknown.

Measure E7

Percentage of children living in areas with major violations of drinking water monitoring and reporting requirements



SOURCE: U.S. Environmental Protection Agency, Office of Water, Safe Drinking Water Information System (Percentages are estimated)

- In 1993, approximately 22 percent of children lived in an area served by a public water system that had at least one major monitoring and reporting violation. This figure decreased to about 10 percent in 1999.
- The largest number of monitoring and reporting violations occurred for the lead and copper standards. Approximately 11 percent of children in 1993 were served by public water systems with monitoring and reporting violations for lead and copper, decreasing to about 5 percent in 1995. The number has remained relatively constant since then.
- The percentage of children living in areas with a major chemical and radiation monitoring violation declined from approximately 8 percent in 1993 to about 3 percent in 1999.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants: lead (E1-E2) *Drinking Water: lead (E6-E7)*	Lead in Blood (B1-B3)	Neurodevelopmental (D7)	Lead in Schools (S1-S3)

Pesticide Residues on Foods Frequently Consumed by Children

Children may be exposed to pesticides and other contaminants in their food and through day-to-day activities around the home. EPA regulates the amounts of pesticides in food, termed "residues," through standards called "food tolerances." A tolerance is a legal limit on the amount of pesticide residue in a particular food.

Children's exposures to pesticides may be higher than the exposures of most adults. Pound for pound, children generally eat more than adults, and they may be exposed more heavily to certain pesticides because they consume a diet different from that of adults.[87] Among the agricultural commodities that are consumed by children in large amounts are apples, corn, oranges, rice, and wheat.

Organophosphate pesticides frequently are applied to many of the foods important in children's diets, and certain organophosphate pesticide residues can be detected in small quantities. When exposure to organophosphate pesticides is sufficiently high, they interfere with the proper functioning of the nervous system.[88] There are approximately 40 organophosphates, and as a group they account for approximately half of the insecticide use in the United States. The majority of organophosphate use is on food crops—including corn, fruits, vegetables, and nuts. In addition, organophosphate pesticides often have been used in and around the home. Examples of organophosphate pesticides include chlorpyrifos, azinphos methyl, methyl parathion, and phosmet.

The U.S. Department of Agriculture (USDA) collects annual data on pesticide residues in food. Among the foods sampled by the USDA's Pesticide Data Program in recent years are several that are important parts of children's diets, including apples, apple juice, bananas, carrots, green beans, orange juice, peaches, pears, potatoes, and tomatoes.

The chart on the following page displays the percentage of food samples with detectable organophosphate pesticide residues reported by the Pesticide Data Program from 1994 to 2001. The 34 organophosphates that were sampled in each of these years are included; other organophosphates that have been added to the program in recent years are excluded so that the chart represents a consistent set of pesticides for all years shown. This measure is a surrogate for children's exposure to pesticides in foods: If the frequency of detectable levels of pesticides in foods decreases, it is likely that exposures will decrease. However, this measure does not account for many additional factors that affect the risk to children. For example, some organophosphates pose greater risks to children than others do, and residues on some foods may pose greater risks than residues on other foods due to differences in amounts consumed. In addition, year-to-year changes in the percentage of samples with detectable pesticide residues may be affected by changes in the selection of foods that are sampled each year.

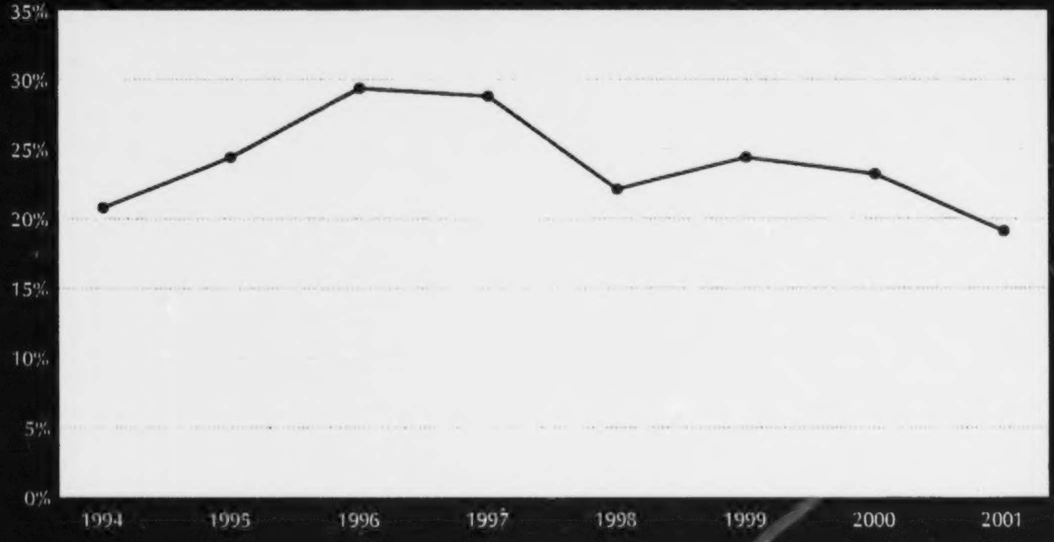
In accordance with the Food Quality Protection Act (FQPA) of 1996, EPA currently is reassessing all food tolerances to assure that they comply with the FQPA's "reasonable certainty of no harm" standard, with a particular focus on protecting children's health. EPA has concluded that a substantial portion of the existing tolerances for organophosphate pesticides meet the stringent safety standards of the FQPA and that a significant portion of the potential exposure to organophosphate pesticides is associated with only a small number of uses of these compounds.

Healthy People 2010: Objective 8-24 of Healthy People 2010 addresses exposure to common pesticides. See Appendix C for more information.

Pesticide Residues

Measure E8

Percentage of fruits, vegetables, and grains with detectable residues of organophosphate pesticides



SOURCE: U.S. Department of Agriculture, Pesticide Data Program

- Between 1994 and 2001, the percentage of food samples with detectable organophosphate pesticide residues ranged between 19 percent and 29 percent. The highest detection rates were observed during 1996 and 1997, while the lowest detection rate was observed in 2001.
- Between 1993 and 2001, the amount of organophosphate pesticides used on foods most frequently consumed by children declined by 44 percent, from 25 million pounds to 14 million pounds.[89]
- In 1999-2000, EPA imposed new restrictions on the use of the organophosphate pesticides azinphos methyl, chlorpyrifos, and methyl parathion on certain food crops and around the home, due largely to concerns about potential exposures of children.

Related Measures:	Environmental Contaminants	Body Burdens	Childhood Illnesses	Special Features
	Pesticide Residues (E8)			*Pesticide Use in Schools (S4)*

Hazardous Waste Sites

Abandoned and uncontrolled hazardous waste sites may pose risks to children who play in or near them.[90,91] These sites also may cause pollution of drinking water, ambient air, and foods. Superfund is the federal government's program to clean up hazardous sites. EPA's principal mechanism for placing sites on Superfund's National Priorities List (NPL) is a scoring system that uses information from initial, limited investigations to assess the relative potential of sites to pose a threat to human health or the environment.

Sites with scores indicating a high risk potential are proposed for addition to the NPL. EPA then accepts public comments on sites, responds to comments, and finalizes the listing for those sites that continue to meet the requirements for addition to the list. Sites on the NPL are studied in detail and cleaned up to the extent necessary to protect human health and the environment. Cleanup has been completed at more than 250 of approximately 1,500 sites to date, and EPA has removed those sites from the NPL. From 1990-2000, 294 sites were added to the NPL. Because the addition and subtraction of sites on the NPL is a continuous process, the number of sites on the NPL stayed relatively constant during this period. The removal of sites from the NPL indicates a decreased risk of exposure to hazardous contaminants. Conversely, the addition of sites to the NPL in recent years does not necessarily reflect an increase in hazards to children. Most of the newly listed sites have been contaminated for many years, many have had exposure restrictions (e.g., fences, or partial cleanups), and their addition to the NPL in the 1990s means only that EPA has recognized the contamination and that the administrative processes required for listing have been completed.

Sites at which substantial cleanup work has been completed may be designated as having reached "Construction Completion." This means that any physical construction necessary to reduce potential exposures has been completed, and other controls are in place to prevent exposure while final cleanup levels are being achieved. Sites with controlled pollution sources represent a level of site remediation at which potential exposures have been significantly reduced, although additional cleanup work remains.

Residence within a mile of a Superfund site is a surrogate measure for exposure to contaminants found at these sites. This measure covers the entire nation and includes data for multiple years. However, residence near a hazardous waste site does not directly represent risks of adverse health effects; the hazards posed to children may vary significantly across the different Superfund sites. In particular, sites that have been controlled (those that have reached Construction Completion) are less likely to pose a hazard than those that are uncontrolled. Some children living near an uncontrolled Superfund site may have relatively low exposure to contaminants originating from that site, while others may have high exposure. This surrogate measure does not imply any specific relationship between childhood illness and a child's proximity to a Superfund site.

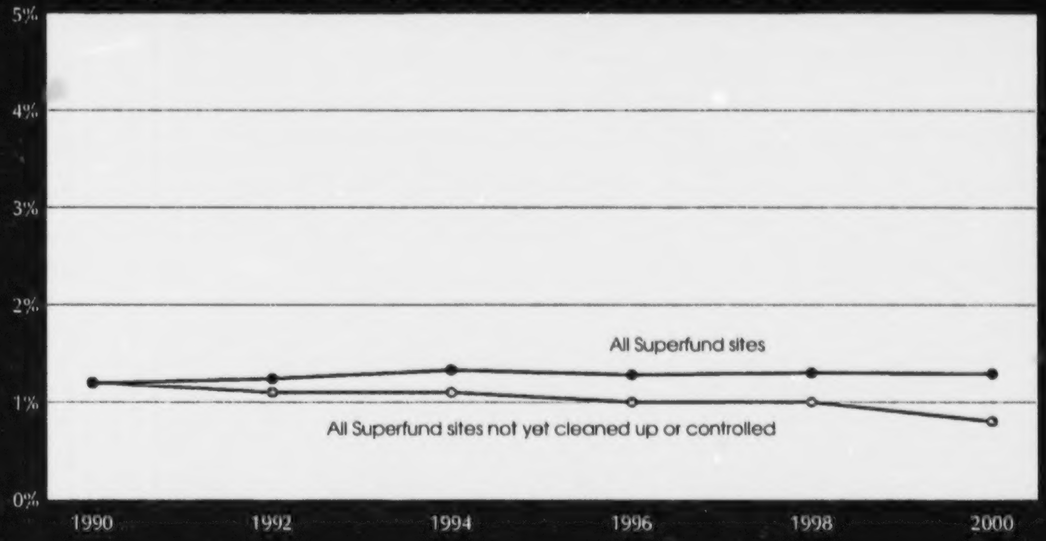
This measure may underestimate the number of children living near hazardous waste sites, since many hazardous sites are not included on the Superfund NPL. For example, the NPL does not include contaminated sites managed by states or addressed as part of the Resource Conservation and Recovery Act corrective action program. Also, this measure most likely underestimates the number of children residing within one mile of a Superfund site, as each site is represented by a single point even though many sites are spread over large areas.

Healthy People 2010: Objective 8-12 of Healthy People 2010 addresses the mitigation of hazardous waste sites on the National Priority List. See Appendix C for more information.

Land Contaminants

Measure E9

Percentage of children residing within one mile of a Superfund site



SOURCE: U.S. Environmental Protection Agency, Superfund NPL Assessment Program (SNAP) Database

- As of September 2000, about 0.8 percent of children lived within one mile of a Superfund site listed on the National Priorities List (NPL) that had not yet been cleaned up or controlled, down from about 1.3 percent in 1990. As of September 2000, about 1.3 percent of children lived within one mile of any Superfund site listed on the Superfund NPL.
- More than 750 out of the approximately 1,500 sites on Superfund's NPL have reached Construction Completion, indicating that potential for exposure has been significantly reduced and controlled. For these sites, any physical construction necessary to reduce potential exposures has been completed, and other controls are in place to prevent exposure while final cleanup levels are being achieved. Final cleanup has been completed at more than 250 of these sites and they have been removed from the NPL.
- As of 2000, approximately 500,000 children lived within one mile of a Superfund site that had been cleaned up or controlled since 1990.

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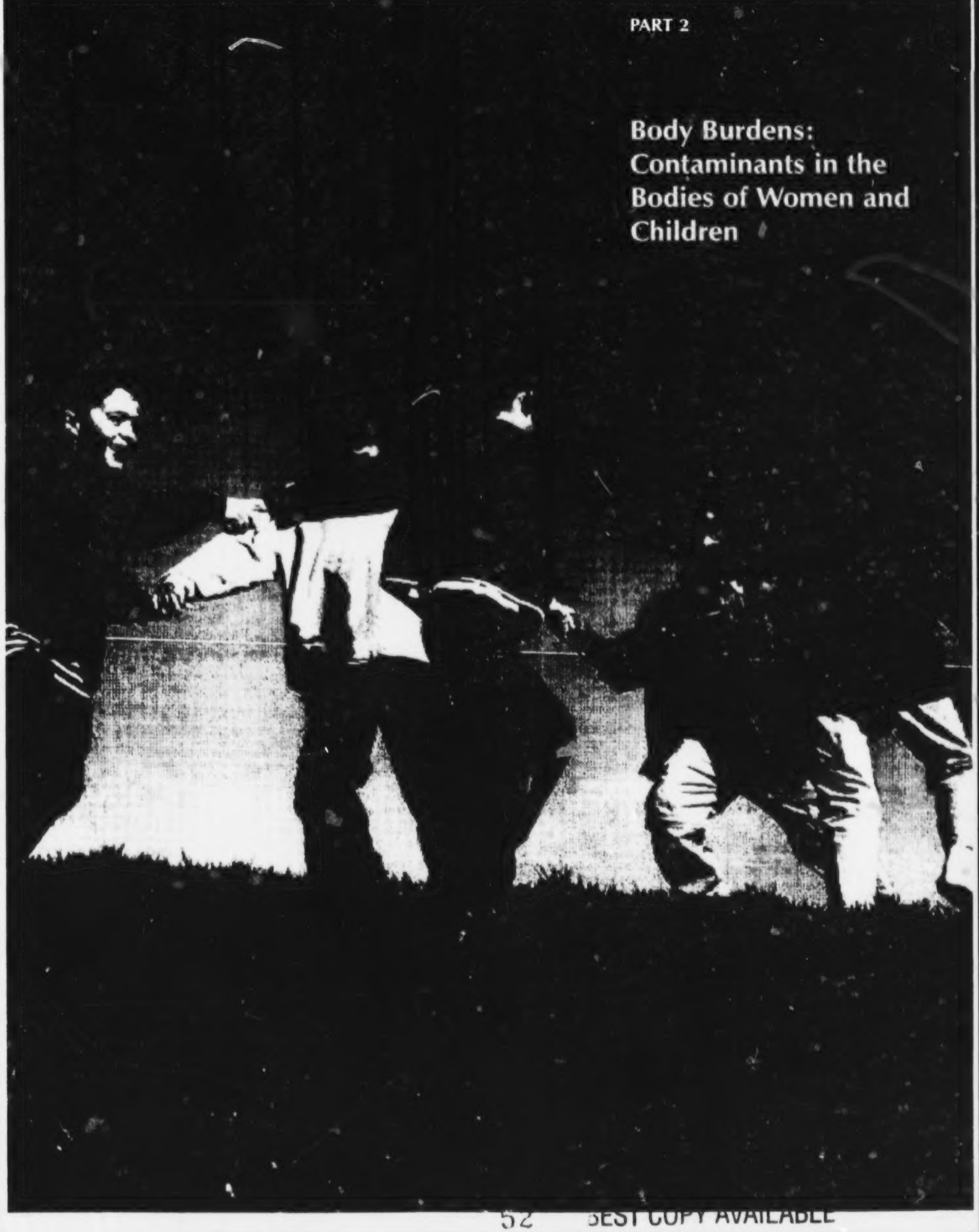
PART 2

Body Burdens: Contaminants in the Bodies of Women and Children



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Body Burdens

Measurements of the levels of pollutants in children's bodies provide direct information about exposures to environmental contaminants. Also, measurements in women who may become pregnant, currently are pregnant, or currently are breastfeeding provide information about exposures that potentially can affect conception, the fetus, and/or the developing child. These "body burden" measurements most often are taken from blood samples, but also can come from urine or hair.

This edition of *America's Children and the Environment* includes new body burden measurements for mercury and cotinine. The first new measure shows concentrations of mercury in the blood of women of child-bearing age, defined as between the ages of 16 and 49. This measure is important because studies have shown that prenatal exposure to mercury can cause adverse neurological and developmental effects in children.

The second new measure shows levels of cotinine in the blood of children. Cotinine is a breakdown product of nicotine. Children who have been exposed to environmental tobacco smoke (ETS) have cotinine in their blood. Exposure to ETS increases the risk of a number of adverse health effects, including lower respiratory tract infections, bronchitis, pneumonia, ear infections, asthma symptoms, and sudden infant death syndrome (SIDS).

This report also includes updated data on lead in the blood of children. This measure is directly related to adverse neurological and developmental effects in children. The data on concentrations of lead in blood (called "blood lead") depict a trend over 25 years.

One limitation of body burden measures is that they reveal few clues to the source(s) of exposure. For example, lead in children's blood may come from exposure to airborne sources, contaminated water or food, or contaminated soil or dust.

The measures in this section do not account for many environmental contaminants that are important to children but for which data are not available on a national scale, or for which information is lacking to evaluate health significance. For example, data are now available for a number of other environmental contaminants—such as pesticides and heavy metals—in children's blood and urine. However, no information is available to show how these concentrations relate to health risks. Also, it is not currently possible to show trends for these contaminants because data are available for only one year.

Concentrations of Lead in Blood

Lead in the Blood of Children

Lead is a major environmental health hazard for young children. Childhood exposure to lead contributes to learning problems such as reduced intelligence and cognitive development.[1-3] Studies also have found that childhood exposure to lead contributes to attention-deficit/hyperactivity disorder[4] and hyperactivity and distractibility;[5-7] increases the likelihood of dropping out of high school, having a reading disability, lower vocabulary, and lower class standing in high school;[8] and increases the risk for antisocial and delinquent behavior.[9] A blood lead level of 10 micrograms per deciliter (µg/dL) or greater is considered elevated,[10, 11] but there is no demonstrated safe concentration of lead in blood.[12] Adverse health effects can occur at lower concentrations.[2, 13, 14]

In the past, ambient concentrations of lead from leaded gasoline were a major contributor to blood lead levels in children.[14] Today, elevated blood lead levels are due mostly to ingestion of contaminated dust, paint and soil.[10] Soil and dust that are contaminated with lead are important sources of exposure because children play outside, and very small children frequently put their hands in their mouths.[15, 17] Deterioration of lead-based paint can generate contaminated dust and soil, and past emissions of lead in gasoline that subsequently were deposited in the soil also contribute to lead-contaminated soil and house dust.[15-17] As of 1998-2000, lead-based paint was present in 40 percent of U.S. homes.[18] 16 percent of homes had dust lead hazards, and 7 percent of homes had soil lead hazards.[18] Some small fraction of children also are exposed through direct ingestion of lead-containing paint chips.[19]

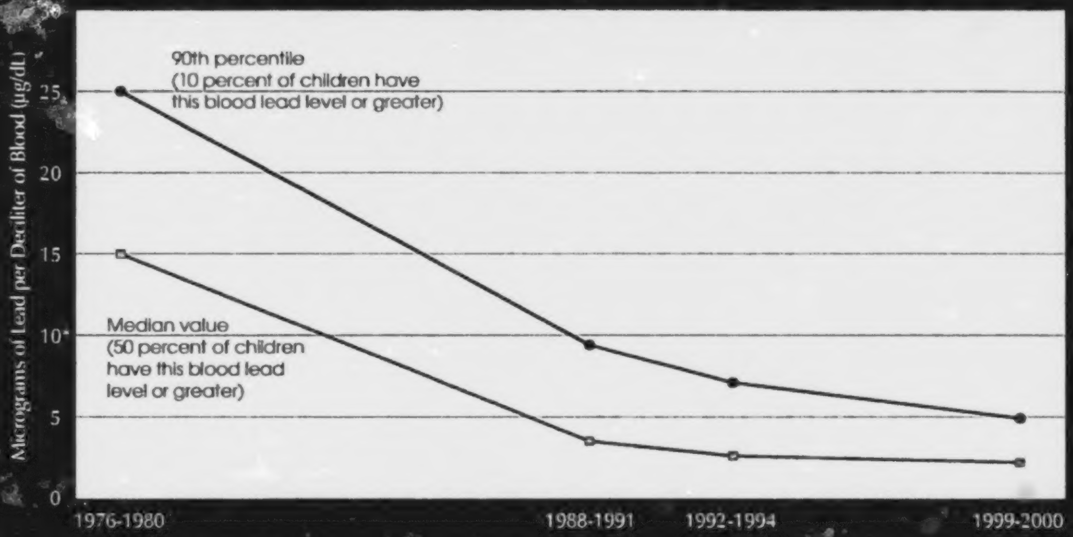
Although the concentration of lead in blood is an important indicator of risk, it reflects only current exposures. Lead also accumulates in bone. Recent research suggests that concentrations of lead in bone may be more related to adverse health outcomes in children than are concentrations in blood.[20] This finding suggests that concentrations in bone may better reflect the net burden of exposure. However, methods for measuring lead in bone are more time-consuming and expensive than those for measuring lead in blood, and nationally representative data are not available.

Healthy People 2010: Objective 8-11 of Healthy People 2010 aims to totally eliminate elevated blood lead levels in children. See Appendix C for more information.

Concentrations of Lead in Blood

Measure B1

Concentration of lead in blood of children ages 5 and under



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

- The median concentration of lead in the blood of children 5 years old and under dropped from 15 micrograms per deciliter (µg/dL) in 1976-1980 to 2.2 µg/dL in 1999-2000, a decline of 85 percent.
- The concentration of lead in blood at the 90th percentile in children 5 years old and under dropped from 25 µg/dL in 1976-1980 to 4.8 µg/dL in 1999-2000.
- In 1978, about 4.7 million children ages 1-5 had blood lead levels at or greater than 10 µg/dL, which is considered elevated. By 1999-2000, this number had declined to about 300,000.
- The decline in blood lead levels is due largely to the phasing out of lead in gasoline between 1973 and 1995[21] and to the reduction in the number of homes with lead-based paint from 64 million in 1990 to 38 million in 2000.[18] Some decline also was a result of EPA regulations reducing lead levels in drinking water, as well as legislation banning lead from paint and restricting the content of lead in solder, faucets, pipes, and plumbing. Lead also has been eliminated or reduced in food and beverage containers and ceramic ware, and in products such as toys, mini-blinds, and playground equipment.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants: lead (E1-E2) Drinking Water: lead (E6-E7)	*Lead in Blood (B1-B3)*	Neurodevelopmental (D7)	Lead in Schools (S1-S3)

* 10 µg/dL of blood lead has been identified by CDC as elevated, which indicates need for intervention.[22] There is no demonstrated safe concentration of lead in blood.[12] Adverse health effects can occur at lower concentrations.[2,13,14]

Blood Lead by Race/Ethnicity and Family Income

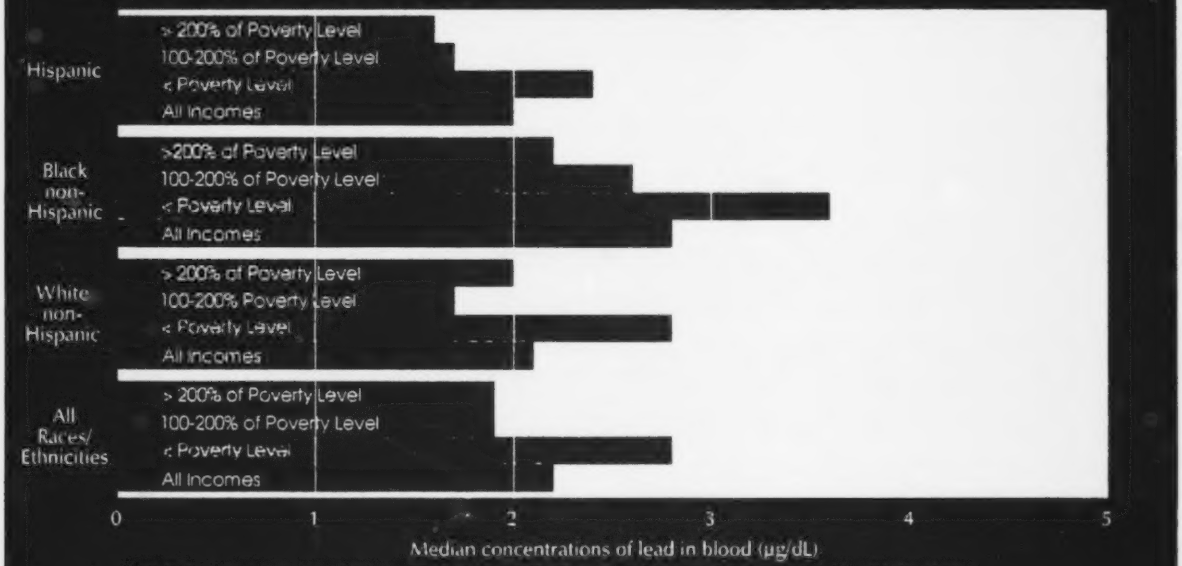
Concentrations of lead in children's blood differ by race/ethnicity and family income. This measure presents blood lead levels by race/ethnicity and family income for children ages 1-5, a period when lead is particularly harmful to the developing brain and nervous system.[22] Measures of blood lead by race/ethnicity and income can help identify the groups that are at greatest risk.

Blood lead levels are highest for younger children, because their exposure per pound of body weight is greater due to their smaller size. In addition, young children play in certain ways that can increase their exposure to lead. For example, they play and crawl on the ground and frequently put their hands in their mouths. Blood lead levels tend to reflect more recent exposures.

Concentrations of Lead in Blood

Measure B2

Median concentrations of lead in blood of children ages 1-5, by race/ethnicity and family income, 1999-2000



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

- In 1999-2000 the median blood lead level in children ages 1-5 was 2.2 µg/dL. The median blood lead level for children living in families with incomes below the poverty level was 2.8 µg/dL and for children living in families above the poverty level it was 1.9 µg/dL.
- In 1999-2000, White non-Hispanic children ages 1-5 had a median blood lead level of about 2 µg/dL, unchanged from the level in 1992-1994.
- In 1992-1994, Black non-Hispanic children ages 1-5 had a median blood lead level of 3.9 µg/dL and in 1999-2000 they had a median blood lead level of 2.8 µg/dL.
- In 1992-1994, Hispanic children ages 1-5 had a median blood lead level of 2.6 µg/dL and in 1999-2000 they had a median blood lead level of 2.0 µg/dL.

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Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants: lead (E1-E2) Drinking Water: lead (E6-E7)	*Lead in Blood (B1-B3)*	Neurodevelopmental (D7)	Lead in Schools (S1-S3)

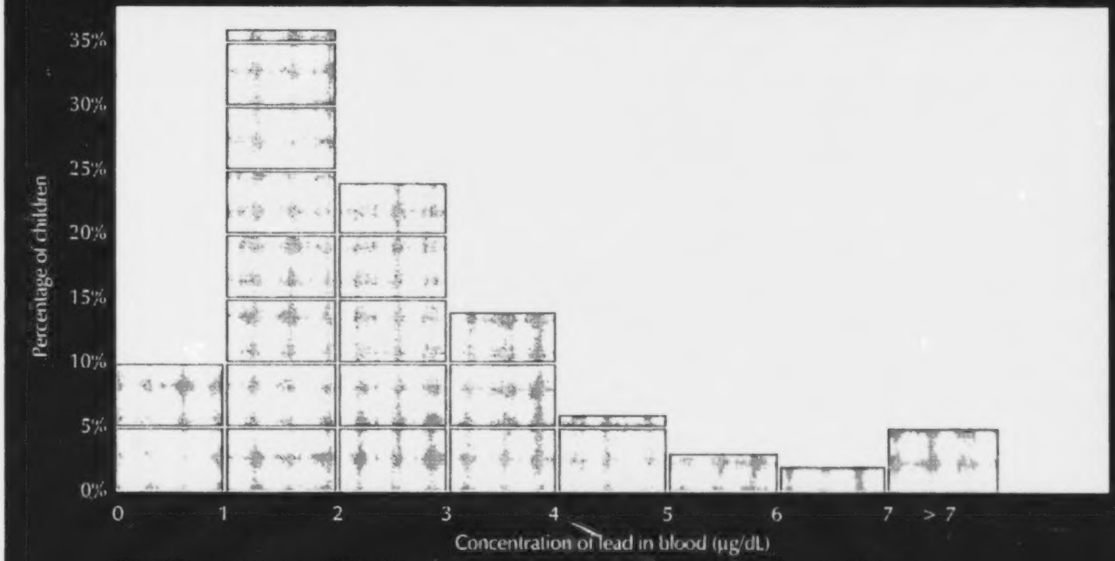
Distribution of Concentrations of Lead in Blood in Children Ages 1-5

A blood lead level of 10 micrograms per deciliter (µg/dL) or greater is considered elevated, but there is no demonstrated safe concentration of lead in blood. Adverse health effects can occur at lower concentrations. A growing body of research has shown that there are measurable adverse neurological effects in children at blood lead concentrations as low as 1 µg/dL.[2, 13, 23] EPA believes that effects may occur at blood lead levels so low that there is essentially no "safe" level of lead.[12] This measure shows the distribution of blood lead levels among children ages 1-5 for the years 1999-2000, the most current years for which data are available.

Concentrations of Lead in Blood

Measure B3

Distribution of concentrations of lead in blood of children ages 1-5, 1999-2000



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

- In 1999-2000, the concentration of lead in blood at the 90th percentile in children ages 1-5 was 4.8 µg/dL, meaning that 10 percent of children had blood lead levels above this concentration and 90 percent had blood lead levels below it.
- In 1999-2000, the median blood lead level of children ages 1-5 was 2.2 µg/dL, meaning that 50 percent of children had blood lead levels above this concentration and 50 percent had blood lead levels below it.
- Approximately 300,000 children ages 1-5 (about 2 percent) had a blood lead level of 10 µg/dL or greater in 1999-2000.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants: lead (E1-E2) Drinking Water: lead (E6-E7)	*Lead in Blood (B1-B3)*	Neurodevelopmental (D7)	Lead in Schools (S1-S3)

Prenatal Exposure to Methylmercury

Prenatal exposure to methylmercury can cause adverse developmental and cognitive effects in children, even at low doses that do not result in effects in the mother.[24-26] Infants and children are particularly sensitive to the effects of neurotoxic agents such as methylmercury.[27] Children who are exposed to low concentrations of methylmercury prenatally are at increased risk of poor performance on neurobehavioral tests, such as those measuring attention, fine motor function, language skills, visual-spatial abilities (like drawing), and verbal memory.[26, 28] There is some evidence that exposure to methylmercury also can affect the cardiovascular,[29] immune,[30, 31] and reproductive systems.[32]

The measure presented here shows blood mercury concentrations in women of child-bearing age (16-49 years). Monitoring the concentrations of mercury in the blood of women of child-bearing age can help identify the proportion of children who may be at risk. Based on recent studies of children born to women who consumed fish that contained mercury, EPA has determined that children born to women with blood mercury concentrations above 5.8 parts per billion are at some increased risk of adverse health effects.[33] EPA's reference dose (RfD) for methylmercury is 0.1 micrograms per kilogram body weight per day. This dose is approximately equal to a concentration of 5.8 parts per billion mercury in blood. Although the prenatal period is the most sensitive period of exposure, exposure to mercury during childhood also could pose a potential health risk.[32]

People are exposed to methylmercury mainly through eating fish contaminated with methylmercury. Mercury that ends up in fish may originate as emissions to the air. Mercury released into the atmosphere can travel long distances on global air currents and be deposited in areas far from its original source.[35, 36] The largest human-generated source of mercury emissions in the United States is the burning of coal. Other sources include the combustion of waste and industrial processes that use mercury.[36]

Mercury usually is released in an elemental form and later converted into methylmercury by bacteria. Methylmercury is more toxic to humans than other forms of mercury, in part because it is more easily absorbed in the body.[36] Methylmercury accumulates through the food chain: fish that live a long time and that eat other fish can accumulate high levels of methylmercury.

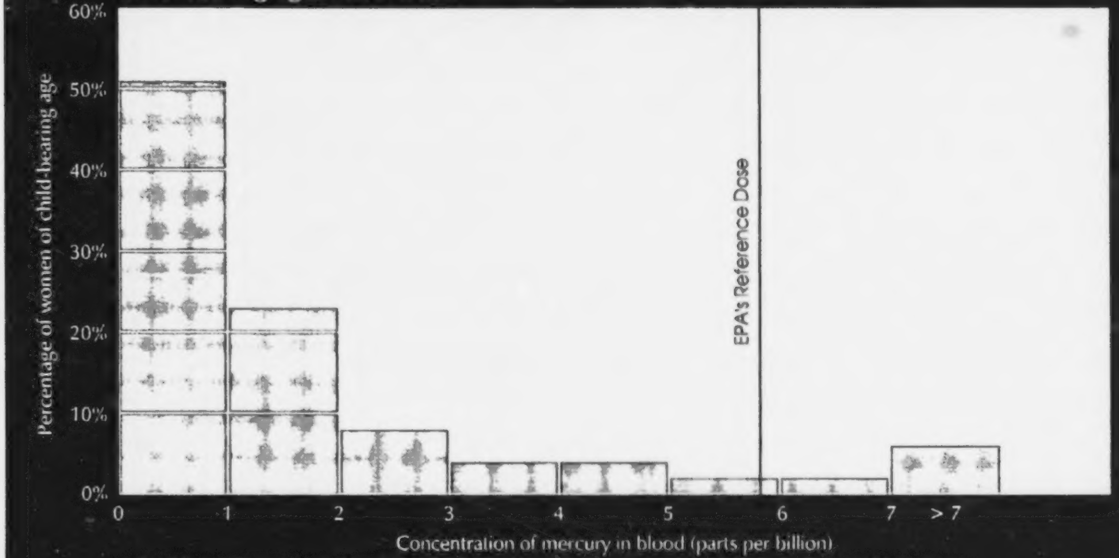
People also can be exposed to inorganic mercury at work, through ritualistic uses of mercury, and from dental restorations with mercury-silver amalgams.[36] Inorganic mercury is less readily absorbed than methylmercury and is not known to cause the types of health effects discussed in this section.

Healthy People 2010: Objective 8-10 of Healthy People 2010 addresses the reduction of contaminants (such as mercury) in fish. See Appendix C for more information.

Concentrations of Mercury in Blood

Measure B4

Distribution of concentrations of mercury in blood of women of child-bearing age, 1999-2000



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

Note: EPA's reference dose (RfD) for methylmercury is 0.1 micrograms per kilogram body weight per day. This is approximately equal to a concentration of 5.8 parts per billion mercury in blood.

- EPA has determined that children born to women with blood concentrations of mercury above 5.8 parts per billion are at some increased risk of adverse health effects.[33] About 8 percent of women of child-bearing age had at least 5.8 parts per billion of mercury in their blood in 1999-2000.
- Current research indicates that there is no safe level of methylmercury in the blood within the range of exposures measured in the human studies of the health effects of mercury, which were as low as 1 part per billion.[33] About 50 percent of the women of child-bearing age in the United States have at least 1 part per billion of mercury in their blood.
- The graph shows reported concentrations of mercury in blood from the National Health and Nutrition Examination Survey. These figures are for total mercury, which includes methylmercury and other forms of mercury. However, most of the mercury in the blood of participants in the survey was methylmercury, so the measured concentrations are a good indication of methylmercury concentrations.[37]
- The National Academy of Sciences (NAS) and EPA have determined that 58 parts per billion of mercury in the blood of pregnant women corresponds to approximately a doubling in the risk of poor performance on a specific neurodevelopmental test.[32-34] The NAS and EPA also have concluded that 32 parts per billion of mercury in the blood of pregnant women corresponds to approximately a doubling in the risk of abnormal performance on a range of neurodevelopmental tests.[32-34] Data from the National Health and Nutrition Examination Survey showed no measured blood mercury concentrations greater than or equal to 58 parts per billion in women of child-bearing age. A small percentage of women (less than 1 percent) have blood mercury concentrations greater than 30 parts per billion.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
		Mercury in Blood (B4)	Neurodevelopmental (D7)	

Concentrations of Cotinine in Blood

Cotinine in the Blood of Children

Exposure to environmental tobacco smoke (ETS) is an important health risk for children. Children who are exposed to environmental tobacco smoke are at increased risk for a number of adverse health outcomes, including lower respiratory tract infections, bronchitis, pneumonia, fluid in the middle ear, and sudden infant death syndrome (SIDS).[38-40] ETS also can play a role in the development and exacerbation of asthma, particularly for children under 6 years old.[41-46] Young children appear to be more susceptible to the effects of environmental tobacco smoke than are older children.[40, 44]

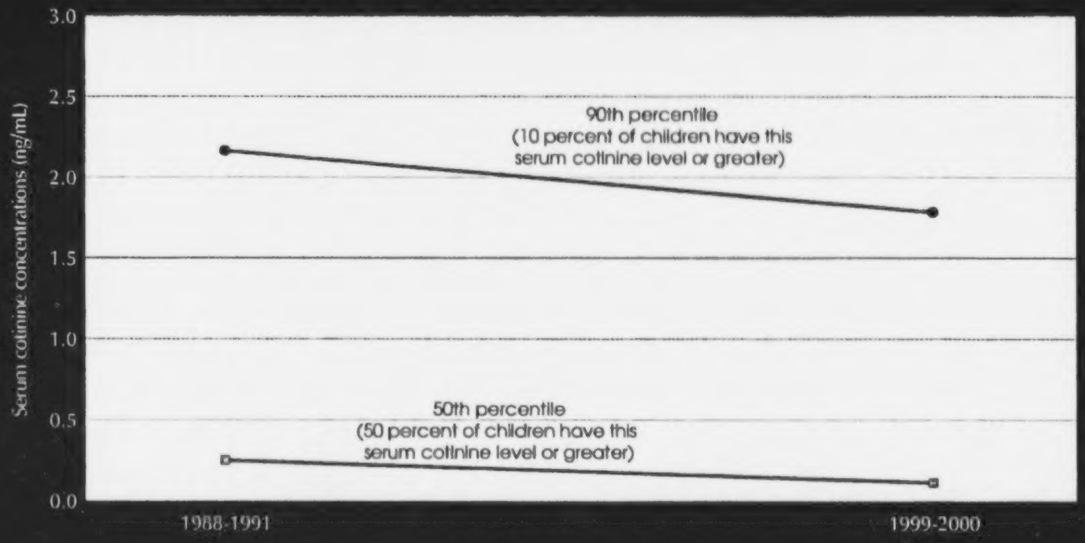
Cotinine is a breakdown product of nicotine in blood. Measurements of cotinine in blood serum are a marker for exposure to environmental tobacco smoke in the previous 1 to 2 days.[47] Children can be exposed to ETS in their homes or in places where people are allowed to smoke, such as some restaurants. This measure presents cotinine levels for non-tobacco-users only. Children who smoke were excluded from these statistics.

Healthy People 2010: Objective 27-9 of Healthy People 2010 is to reduce the proportion of children who are regularly exposed to tobacco smoke at home. See Appendix C for more information.

Concentrations of Cotinine in Blood

Measure B5

Concentrations of cotinine in blood of children



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

- In 1999-2000, median (50th percentile) levels of cotinine measured in children were 56 percent lower than they were in 1988-1991.
- Cotinine values at the 90th percentile, representing the most highly exposed 10 percent of children, showed a smaller relative decline (18 percent) from 1988-1991 to 1999-2000.
- Eighty-five percent of children had detectable levels of cotinine in 1988-1991;[41] between 50 and 75 percent of children had detectable levels of cotinine in 1999-2000 (data not shown).
- The reduction in children's cotinine levels is in part attributable to a decline in the percentage of adults who smoke. In 1999, an estimated 23.5 percent of adults were current smokers, down from 25.0 percent in 1993.[48]
- In 1988-91, median concentrations of cotinine in blood were about 0.6 nanograms per milliliter (ng/mL) for Black non-Hispanic children and about 0.2 ng/mL for White non-Hispanic children and Hispanic children.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Environmental Tobacco Smoke (E5)	*Cotinine in Blood (B5)*	Respiratory (D1-D4)	

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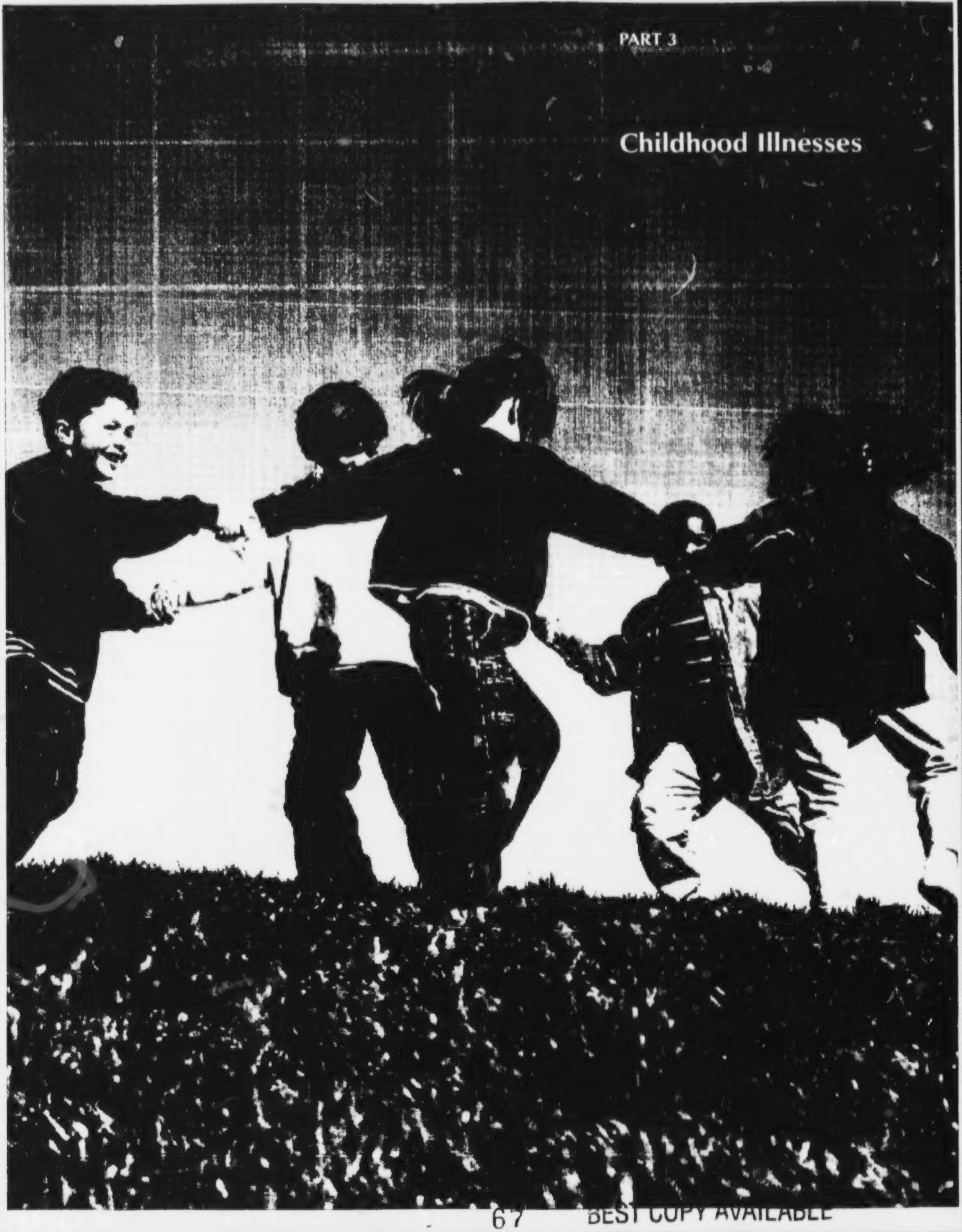
PART 3

Childhood Illnesses



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Childhood Illnesses

Data on trends in childhood diseases and disorders provide important information on successes and shortcomings in efforts to protect children's health. Many important diseases and other health disorders affect children. The causes of many of these conditions are not well established. In some cases environmental contaminants are known to play a role. In other cases clues suggest that environmental factors are important, but definitive proof is lacking.

This section of the report focuses on important childhood diseases and disorders for which evidence or clues indicate or suggest some influence by environmental contaminants, and for which nationally representative data are available. These diseases and disorders are asthma, acute bronchitis and acute upper respiratory infections, cancer, and—new for this edition—neurodevelopmental disorders. Other diseases and disorders that may be influenced partially by environmental contaminants include other respiratory diseases, waterborne diseases, methemoglobinemia, and birth defects.

It is very difficult to develop conclusive evidence that environmental contaminants cause or contribute to the incidence of childhood health effects, particularly those effects occurring in a relatively small proportion of children or effects with multiple causes. In cases where exposure to an environmental contaminant results in a relatively modest increase in the incidence of a disease or disorder, many children would need to be included in a study in order to detect a true relationship. In addition, there may be factors that are related to both the exposure and the health effect (like socioeconomic status) that can make it difficult to detect a relationship between exposure to environmental contaminants and disease. There may, however, be suggestive (rather than conclusive) evidence from studies in humans and/or laboratory animals to suggest that exposures to environmental contaminants contribute to the incidence of a childhood health effect.

Tracking childhood diseases and disorders is an important element of research on potential links between health effects and exposure to environmental contaminants. Tracking establishes a basis for comparison so that increases or decreases in the incidence of a disease or disorder can be detected, often yielding important clues to its causes. Tracking helps researchers determine whether past and current actions have been effective in reducing the incidence of a disease or disorder. It also helps to identify opportunities for further action.

It can be difficult to assess the contribution of environmental exposures to childhood illnesses. Even though environmental exposures can contribute to some childhood illnesses, other factors may be more important, such as family history, nutrition, and socioeconomic factors. In addition, there can be interactions between environmental and genetic factors. This report does not address illnesses that may result from childhood exposures to environmental contaminants but do not manifest themselves until adulthood.

Respiratory Diseases

Respiratory Diseases in Children

Respiratory diseases can greatly impair a child's ability to function, and are an important cause of missed school days and limitations to activities. Important respiratory diseases in children include asthma, bronchitis, and upper respiratory infections.

In 1994-96, 24 percent of children with asthma had to limit their activities due to their asthma, and the disease caused children to miss 14 million days of school.[1] Studies have shown that outdoor and indoor air pollution cause some respiratory symptoms and increase the frequency or severity of asthma attacks.[2-15]

Two types of measures of respiratory diseases are presented here. The first set of measures (D1 and D2) focuses on the percentage of children who have asthma. The second set (D3 and D4) reports on cases of respiratory illness severe enough to require a visit to the emergency room or admission to the hospital.

Asthma

Asthma is a disease of the lungs that can cause wheezing, difficulty in breathing, and chest pain. It is the most common chronic disease among children and is costly in both human and monetary terms.[15]

Asthma varies greatly in severity. Some children who have been diagnosed with asthma may not experience any serious respiratory effects. Other children may have mild symptoms or may respond well to management of their asthma, typically through use of medication. Some children with asthma may suffer serious attacks that greatly limit their activities, result in visits to emergency rooms or hospitals, or, in rare cases, cause death.

Asthma among children is increasing in the United States. Researchers do not understand completely why children develop asthma. The tendency to develop asthma can be inherited, but genetic factors alone are unlikely to explain the significant increases that have occurred in the last 20 years.[15]

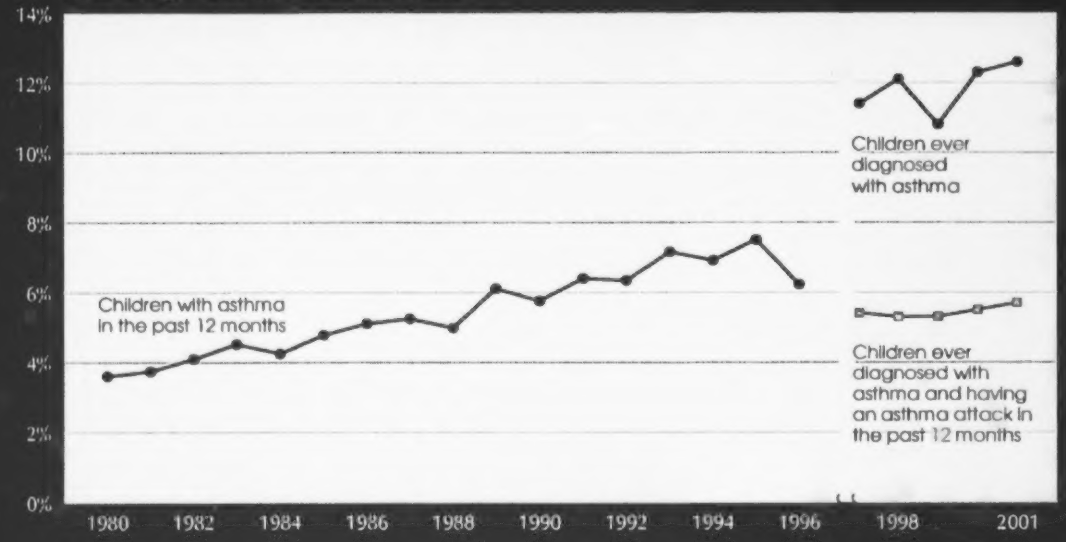
Research on environmental factors that exacerbate or may contribute to causing asthma has focused on environmental agents found outdoors and indoors. The Institute of Medicine concluded that exposure to dust mites causes asthma in susceptible children.[15] Cockroaches and tobacco smoke are likely to cause asthma in young children.[15] Other studies have evaluated the role of indoor air pollutants such as nitrogen dioxide, pesticides, plasticizers, and volatile organic pollutants. Some of these pollutants may play a role in asthma.[15] One recent study suggests that chronic exposure to ozone may be associated with the development of asthma in children who exercise outside,[16] and two other studies suggest that chronic exposure to particulate matter may affect lung function and growth.[17, 18]

Environmental factors may increase the severity or frequency of asthma attacks in children who have the disease. Children with asthma are particularly sensitive to outdoor air pollutants, including ozone, particulate matter, and sulfur dioxide.[2-14, 19] These pollutants can exacerbate asthma, leading to difficulty in breathing, an increased use of medication, visits to doctors' offices, trips to emergency rooms, and admissions to the hospital. In addition, one study reported a relationship between exposure to hazardous air pollutants and increases in chronic respiratory symptoms that are characteristic of asthma.[20]

Data from the National Health Interview Survey were used to estimate the prevalence of childhood asthma. For 1980 to 1996, the percentage of children reported to have asthma in the preceding 12 months is shown. In 1997, the survey's method for measuring childhood asthma changed. For 1997 to 2001, the measure shows the percentage of children who had ever been told by a doctor or health professional that they have asthma, as well as the percentage of children who were ever diagnosed with asthma and who had an asthma attack in the preceding 12 months. Some children may have asthma when they are young and outgrow it as they get older, or their asthma may be well-controlled through medication and by avoiding triggers of asthma attacks. In such cases, children may have asthma but may not have experienced any attacks in a long time.

Measure D1

Percentage of children with asthma



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

Note: The survey questions for asthma changed in 1997; data before 1997 cannot be directly compared to data in 1997 and later.

- Between 1980 and 1995, the percentage of children with asthma doubled, from 3.6 percent in 1980 to 7.5 percent in 1995. A decrease in the percentage of children with asthma occurred between 1995 and 1996, but it is difficult to interpret single-year changes.
- It is difficult to obtain an accurate measurement of how many children have asthma, because asthma is a complex disease that can be difficult to differentiate from other wheezing disorders, especially in children under the age of 6 years. Prior to 1997, the percentage of children with asthma was measured by asking parents if a child in their family had asthma during the previous 12 months. In 1997-2001, a parent was asked if his or her child had ever been diagnosed with asthma by a health professional. If the parent answered yes, then he or she was asked if the child had an asthma attack or episode in the last 12 months. The percentage of children with an asthma attack in the last 12 months measures the population with incomplete control of asthma. For 1997-2001, available data do not distinguish between those children who may no longer have active asthma and those whose asthma is well controlled.
- Starting in 2001, the National Health Interview Survey included a question that allows the estimation of the percentage of children who currently have asthma. The results indicate that 8.7 percent (6.3 million) of children had asthma in 2001.

Related Measures:	Environmental Contaminants	Body Burdens	Childhood Illnesses	Special Features
	Criteria Air Pollutants (E1-E3)		*Respiratory (D1-D4)*	
	Environmental Tobacco Smoke (E5)	Cotinine in Blood (B5)	*Respiratory (D1-D4)*	

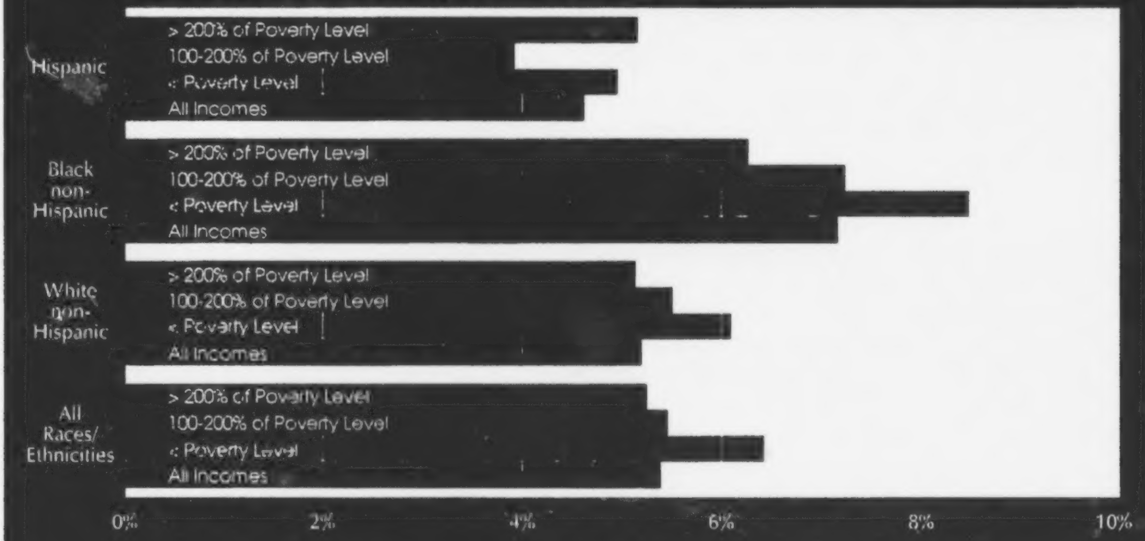
Percentage of Children Having an Asthma Attack in the Previous 12 Months, by Race/Ethnicity and Family Income

Children of lower-income families and children of color are more likely to have had an asthma attack in the previous 12 months. These children may face barriers to medical care, or they may have less access to routine medical care and instructions for asthma management than other children do. These factors can increase the severity and impact of the illness.[21-24] Data for 1997-2000 show that the percentage of children with asthma having an asthma attack in the last 12 months differs by racial and ethnic groups and by family income.

The Institute of Medicine concluded that exposure to dust mites causes asthma in susceptible children.[15] Cockroaches and tobacco smoke are likely to cause asthma in young children.[15] Research suggests that lower income children are more likely to live in homes with higher exposure to cockroach allergens.[25, 26] The first nationally representative survey of allergens in U.S. housing reported higher levels of dust mite allergen in bedding from lower income families.[27] Although some studies found higher dust mite allergen levels in the homes of higher income families, those studies were conducted in smaller geographic areas.[25, 26]

Measure D2

Percentage of children having an asthma attack in the previous 12 months, by race/ethnicity and family income, 1997-2000



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

- In 1997-2000, 5.5 percent of all children had an asthma attack in the previous 12 months.
- More than 8 percent of Black non-Hispanic children living in families with incomes below the poverty level had an asthma attack in the previous 12 months.
- Approximately 6 percent of White non-Hispanic children and 5 percent of Hispanic children living in families with incomes below the poverty level had an asthma attack in the previous 12 months.
- More than 6 percent of children living in families with incomes below the poverty level had an asthma attack in the previous 12 months. About 5 percent of children living in families with incomes at the poverty level and higher had an asthma attack in the previous 12 months.

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Related Measures:	Environmental Contaminants	Body Burdens	Childhood Illnesses	Special Features
	Criteria Air Pollutants (E1-E3)		*Respiratory (D1-D4)*	
	Environmental Tobacco Smoke (E5)	Cotinine in Blood (B5)	*Respiratory (D1-D4)*	

Emergency Room Visits and Hospitalizations for Respiratory Diseases

Children who visit emergency rooms or are hospitalized for respiratory diseases—such as asthma, upper respiratory infections, and acute bronchitis—represent the most severe cases of respiratory effects.

Only a fraction of children with respiratory diseases are admitted to the hospital. Hospital admissions and emergency room visits for respiratory diseases can be related to a number of factors besides air pollution, such as lack of access to primary health care and instructions for asthma management. Changes in hospital admissions and emergency room visits over time may reflect changes in medical practices, asthma therapy, and access to and use of care.[28, 29]

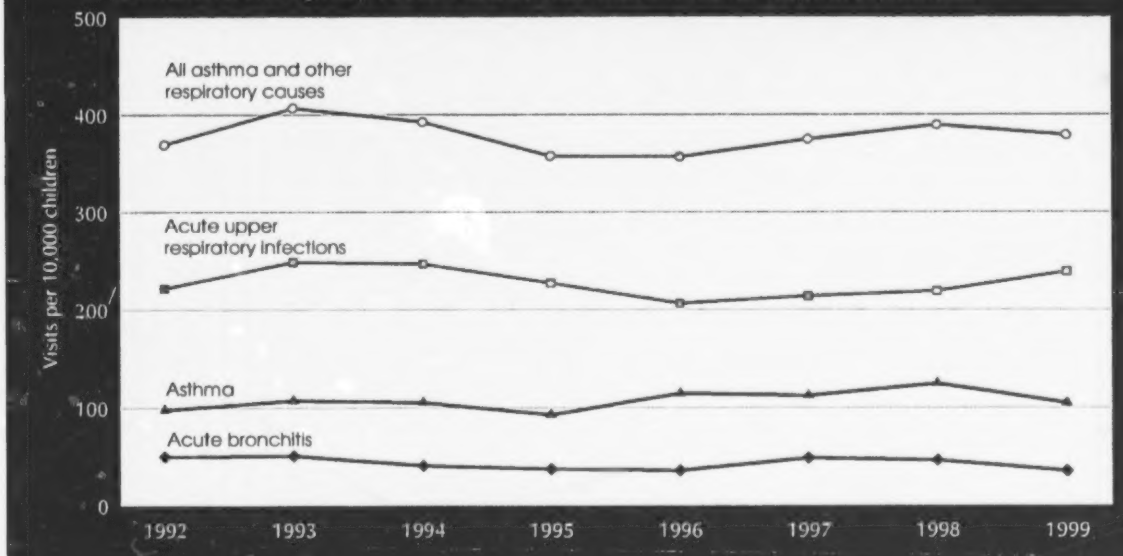
There is extensive scientific evidence that exposure to air pollution from outdoor and indoor sources can exacerbate existing respiratory conditions.[12-14] For children with these conditions, exposure to air pollution can lead to difficulty in breathing, increased use of medication, visits to the doctor's office, trips to the emergency room, and in some cases admission to the hospital.[30-33] For example, outdoor air pollution can cause asthma attacks in children, which can lead to emergency room visits.[8, 9, 34, 35] A recent study found that increased ozone concentrations in the summer were related to increased respiratory-related hospital and emergency room visits for children under the age of two.[19] Studies conducted in the northeastern United States indicate that air pollution during the summer was associated with approximately 6-24 percent of all hospital admissions for asthma.[36]

Recent analyses also have suggested that exacerbation of asthma from exposure to air pollution can be more severe among lower-income people than in other populations.[37, 38]

Respiratory Diseases

Measure D3

Children's emergency room visits for asthma and other respiratory causes



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Hospital Ambulatory Medical Care Survey

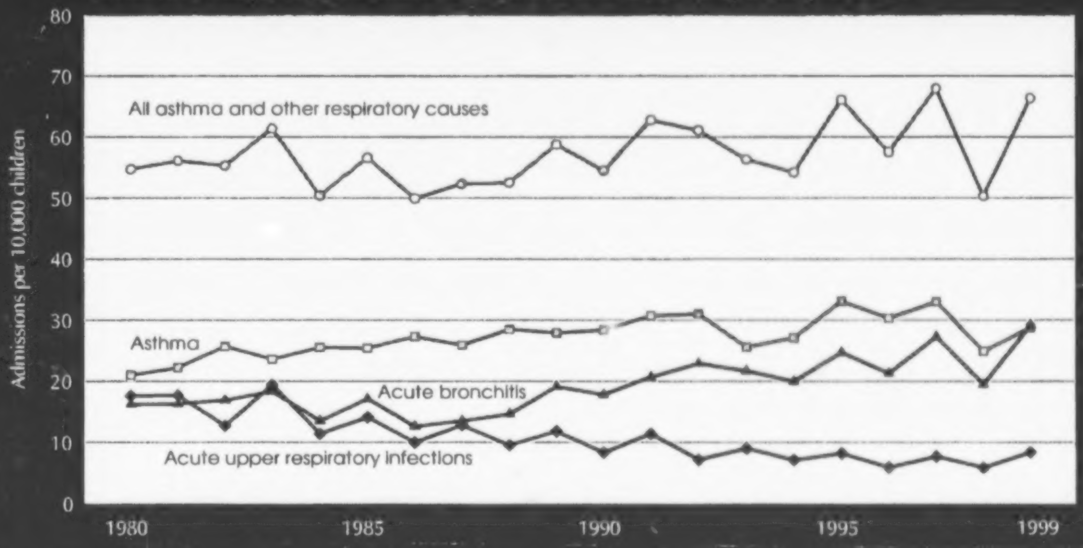
- Emergency room visits for asthma and other respiratory causes were 369 per 10,000 children in 1992 and 379 per 10,000 children in 1999.
- Trends in individual causes of emergency room visits remained fairly stable between 1992 and 1999. In 1999, hospitals reported 239 emergency room visits per 10,000 children for acute upper respiratory infections, 104 visits per 10,000 children for asthma, and 35 visits per 10,000 children for acute bronchitis.
- Data on children's emergency room visits for asthma and other respiratory causes by race and ethnicity are shown in the data tables in Appendix A.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants (E1-E3)		*Respiratory (D1-D4)*	
	Environmental Tobacco Smoke (E5)	Cotinine in Blood (B5)	*Respiratory (D1-D4)*	

Respiratory Diseases

Measure D4

Children's hospital admissions for asthma and other respiratory causes



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Hospital Discharge Survey

- Hospital admissions for asthma and other respiratory causes were 55 per 10,000 children in 1980 and 66 per 10,000 children in 1999.
- Hospital admissions for asthma alone increased from 21 per 10,000 children in 1980 to 29 per 10,000 children in 1999. Hospital admissions for acute bronchitis increased from 16 per 10,000 children in 1980 to 29 per 10,000 children in 1999. Hospital admissions decreased for acute upper respiratory infections from 18 per 10,000 children in 1980 to 8 per 10,000 children in 1999.
- Asthma hospitalizations accounted for about 7 percent of all hospitalizations for children aged 0-14 in 1999, and asthma was the fourth leading cause of non-injury-related hospital admissions in that year.[39]
- Acute bronchitis accounted for about 8 percent of all hospitalizations for children aged 0-14 in 1999. Acute bronchitis was the third leading cause of non-injury related hospital admissions in that year.[39]
- Children aged 0-14 represented 40 percent of asthma hospitalizations for all ages (children and adults) during 1999.[39]
- Children's access to primary and preventive care also plays a role in the number of hospitalizations.
- Data on children's hospital admissions for asthma and other respiratory causes by race and ethnicity are shown in the data tables in Appendix A.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants (E1-E3)		*Respiratory (D1-D4)*	
	Environmental Tobacco Smoke (E5)	Cotinine in Blood (B5)	*Respiratory (D1-D4)*	

Cancer Incidence and Mortality

Cancer in childhood is quite rare compared with cancer in adults, but it still causes more deaths than any factor, other than injuries and accidents, among children 1-19 years of age.[40]

Childhood cancer is not a single disease, but includes a variety of malignancies. The forms of cancer that are most common vary according to age.

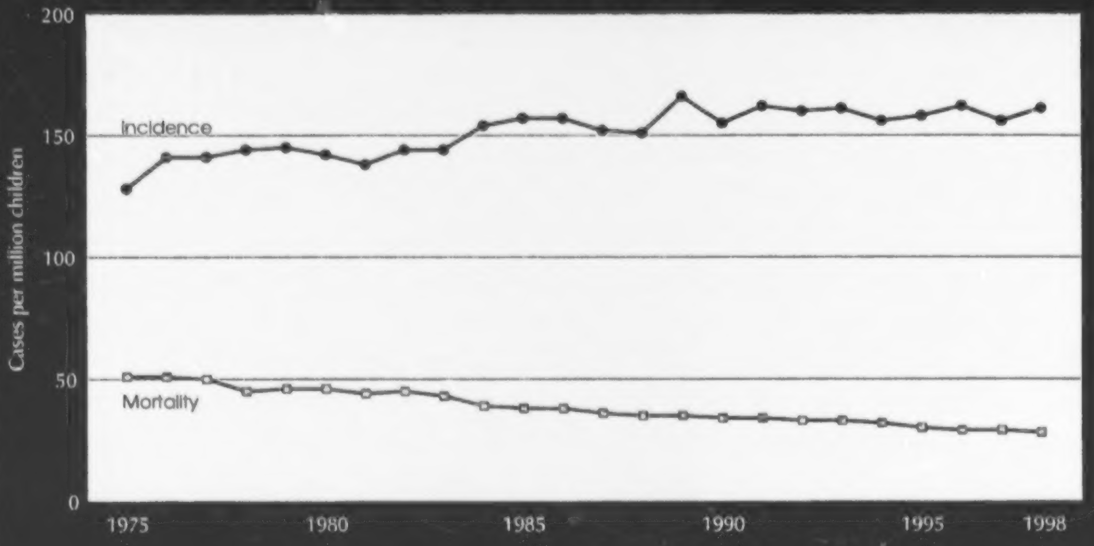
The annual incidence of childhood cancer increased from 1975 until about 1990. The frequency of the disease appears to have become fairly stable overall since 1990. Mortality has declined substantially during the last 25 years, due largely to improvements in treatment.

The causes of cancer in children are poorly understood, though in general it is thought that different forms of cancer have different causes. Established risk factors for the development of childhood cancer include family history, specific genetic syndromes, radiation, and certain pharmaceutical agents used in chemotherapy.[40] Evidence from epidemiological studies suggests that environmental contaminants such as pesticides and certain chemicals, in addition to radiation, may contribute to an increased frequency of some childhood cancers.[40, 41] Some studies have found that children born to parents who work with or use such chemicals are more likely to have cancer in childhood.[40, 42] It may be that the chemicals cause mutations in parents' germ cells that increase the risk of their children developing certain cancers, or perhaps the parental exposure is passed on to the child while *in utero*, affecting the child directly. Children's direct exposures to such chemicals also may contribute to cancer.

Childhood Cancer

Measure D5

Cancer incidence and mortality for children under 20



SOURCE: Incidence data from National Cancer Institute: Surveillance, Epidemiology, and End Results Program; mortality data from Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System

- The frequency of new cancer cases has been fairly stable since 1990. The age-adjusted annual incidence of cancer in children increased from 128 to 161 cases per million children between 1975 and 1998. Cancer mortality decreased from 51 to 28 deaths per million children during the 1975-1998 period.
- Rates of cancer incidence vary by age. Rates are highest among infants, decline until age 9, and then rise again with increasing age. Between 1986 and 1995, children under 5 and those aged 15-19 experienced the highest incidence rates of cancer at approximately 180 cases per million. Children aged 5-9 and 10-14 had lower incidence rates at approximately 100 and 110 cases per million respectively.
- Between 1994 and 1998, incidence rates of cancer were highest among White non-Hispanics at 172 per million for boys and 156 per million for girls. Hispanics were next highest at 150 per million for boys and 141 per million for girls. Asians and Pacific Islanders had an incidence rate of 150 per million for boys and 132 per million for girls. Black non-Hispanic children had a rate of 133 per million for boys and 117 per million for girls. American Indians and Alaska Natives had the lowest rate at 82 per million for boys and 62 per million for girls. Data on childhood cancer incidence and mortality by race and ethnicity are shown in the data tables in Appendix A.

Types of Childhood Cancer

Trends in the total incidence of childhood cancer are useful indicators for assessing the overall burden of cancer among children. However, broad trends mask changes in the frequency of individual cancers. Individual cancers often have patterns that diverge from the overall trend. Moreover, environmental factors may be more likely to contribute to some childhood cancers than to others.

Ionizing radiation, such as from x-rays, is a known cause of leukemia and brain tumors.[43, 44] There is suggestive—but not conclusive—evidence that parental exposures to certain chemicals may be a cause of leukemia, brain cancer, non-Hodgkin's lymphoma, and Wilms' tumor in children.[40, 42, 45]

A number of studies have evaluated the relationship between pesticide exposure and certain types of childhood cancer. Although the evidence is suggestive of a link, it is not conclusive.[40] Most studies of the relationship between pesticide exposure and leukemia and brain cancer show increased risks for children whose parents used pesticides at home or work, as well as for children who may be exposed to pesticides in the home.[46-49] Evidence is limited but suggestive that non-Hodgkin's lymphoma in children may be linked to parental pesticide exposure and exposure to pesticides in the home.[47] There is some evidence linking pesticide use to Wilms' tumor and Ewing's sarcoma.[47]

There is also suggestive, though not conclusive, evidence that maternal and paternal exposure to solvents may increase the risk of childhood cancers. A recent review found that there is strong evidence for an association between paternal exposure to solvents—including benzene, carbon tetrachloride, and trichloroethylene—and childhood leukemias.[42] A number of studies also find a link between childhood cancer and paternal employment in occupations related to motor vehicles or involving exposure to exhaust gas.[42] In addition, a recent study found an association between living close to areas with heavy traffic and childhood leukemia.[50] The authors of these studies suggest that the link in these cases may be benzene, which is associated with leukemia in adults.[42, 50]

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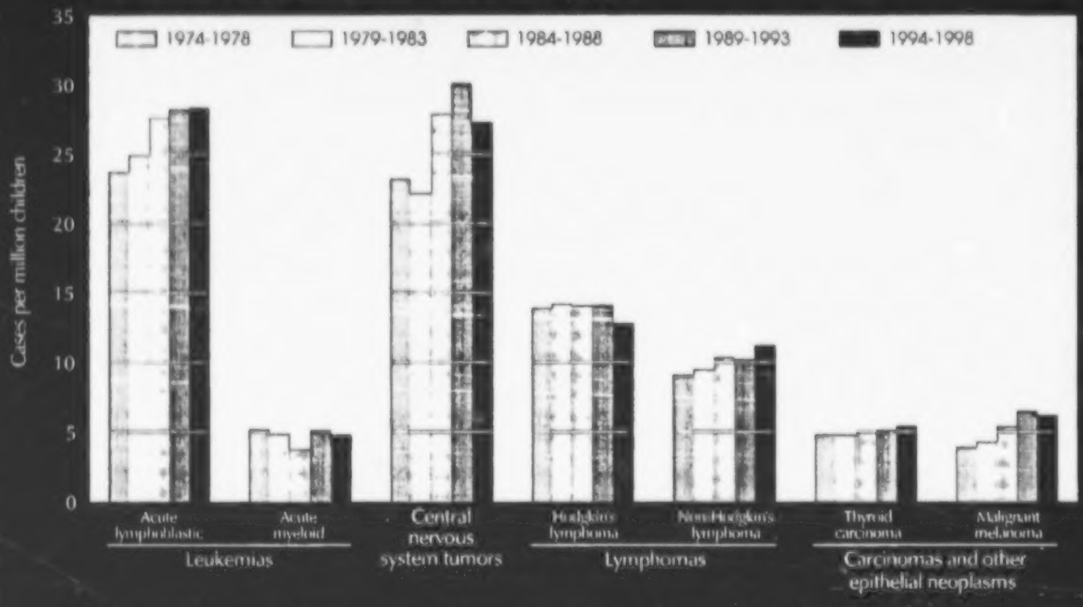
Measure D6a

Cancer incidence for children under 20 by type



SOURCE: National Cancer Institute; Surveillance, Epidemiology, and End Results Program

- Leukemia was the most common cancer diagnosis for children from 1973-1998, representing about 20 percent of total cancer cases. Incidence of acute lymphoblastic leukemia was 24 cases per million in 1974-1978 and approximately 28 cases per million in 1994-1998. Rates of acute myeloid leukemia were approximately 5 cases per million in 1974-98 and about the same in 1994-98.
- Central nervous system tumors represented about 17 percent of childhood cancers. The incidence of central nervous system tumors was approximately 23 cases per million in 1974-1978 and 27 per million in 1994-1998.
- Lymphomas, which include Hodgkin's disease and non-Hodgkin's lymphoma, represent approximately 15 percent of childhood cancers. Incidence of Hodgkin's disease was roughly 14 cases per million in 1974-1978 and 13 per million in 1994-1998. There were approximately 9 cases of non-Hodgkin's lymphomas per million children in 1974-1978 and 11 per million in 1994-1998.

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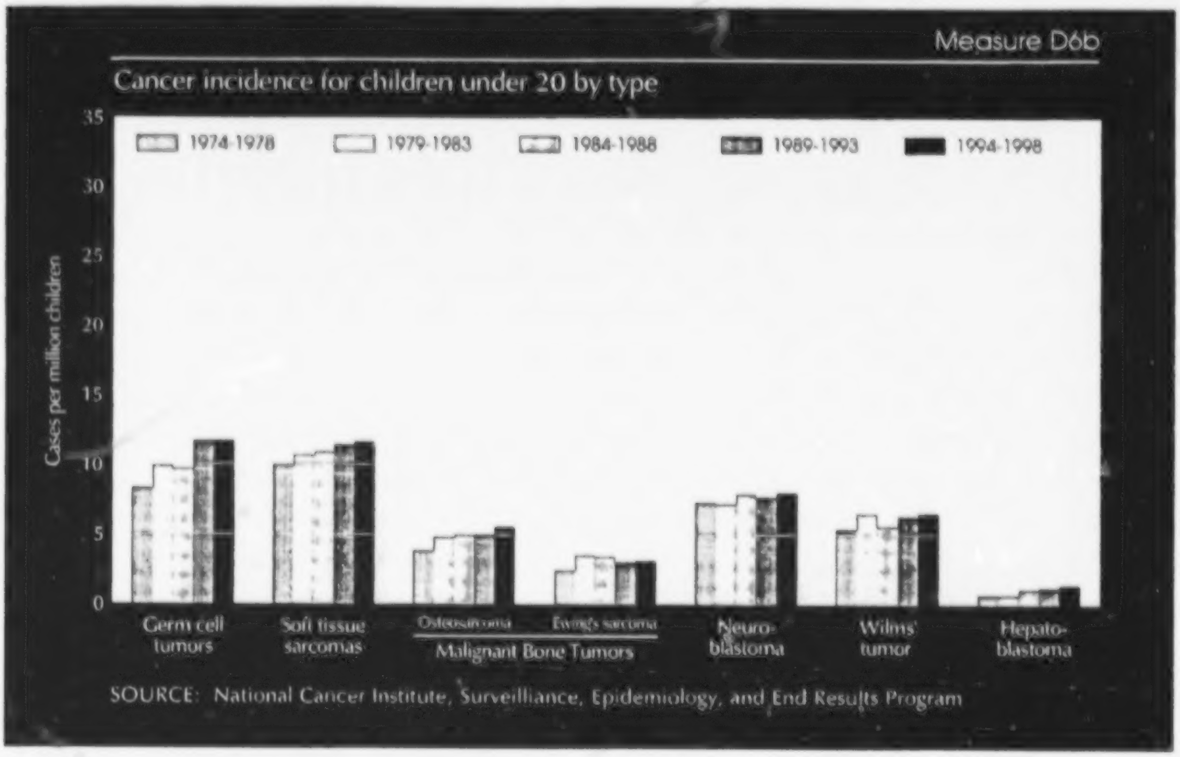


Measure D6b

Cancer incidence for children under 20 by type

SOURCE: National Cancer Institute, Surveillance, Epidemiology, and End Results Program

- Different types of cancer affect children at different ages. Neuroblastomas and Wilms' tumor (tumors of the kidney) are usually found only in very young children. Nervous system cancers and leukemias are most common through age 14 (leukemias being highest among 0-4 year olds); lymphomas, carcinomas, and germ cell and other gonadal tumors are more common in those 15-19 years old.[40]

Neurodevelopmental Disorders in Children

Researchers estimate that between 3 and 8 percent of the babies born in the United States each year will be affected by neurodevelopmental disorders such as attention-deficit/hyperactivity disorder or mental retardation.[51] Neurodevelopmental disorders are disabilities in the functioning of the brain that affect a child's behavior, memory, or ability to learn. These effects may result from exposure of the fetus or young child to certain environmental contaminants, though current data do not indicate the extent to which environmental contaminants contribute to overall rates of neurodevelopmental disorders in children. A child's brain and nervous system are vulnerable to adverse impacts from pollutants because they go through a long developmental process beginning shortly after conception and continuing through adolescence.[52, 53]

Studies have found that several widespread environmental contaminants can damage children's developing brain and nervous system. Childhood exposure to lead contributes to learning problems such as reduced intelligence and cognitive development.[54-56] Studies also have found that childhood exposure to lead contributes to attention-deficit/hyperactivity disorder[57] and hyperactivity and distractibility;[58-60] increases the likelihood of dropping out of high school, having a reading disability, lower vocabulary, and lower class standing in high school;[61] and increases the risk for antisocial and delinquent behavior.[62]

Methylmercury also has negative impacts on children's neurological development. Studies of children whose mothers had high intakes of mercury-contaminated seafood prior to conception found adverse impacts on intelligence[63, 64] and decreased functioning in the areas of language, attention, and memory.[65] Particularly high levels of exposure to mercury in the womb have been found to cause mental retardation.[66, 67]

Several studies of children exposed to elevated levels of polychlorinated biphenyls (PCBs) have linked these contaminants to neurodevelopmental effects, including lowered intelligence and behavioral deficits such as inattention and excessive reaction to stimulation. Most of these studies find that the effects are associated with exposure in the womb resulting from the mother having eaten food contaminated with PCBs.[68-73] Adverse effects on intelligence and behavior also have been found in children of women who were highly exposed to mixtures of PCBs, chlorinated dibenzofurans, and other pollutants prior to conception.[74-76]

Human studies also suggest that exposures to other metals such as cadmium and arsenic may have adverse effects on neurological development.[58, 77-79] Other types of pollutants also have been associated in animal studies with neurodevelopmental effects. Numerous toxicological studies link both prenatal and postnatal exposure to organophosphate pesticides to neurodevelopmental effects.[80] A recent study of brominated flame retardants found that two of these compounds caused adverse effects on behavior, learning, and memory in animals.[81]

Healthy People 2010: Objective 16-14 of Healthy People 2010 calls for a reduction in the occurrence of developmental disabilities. See Appendix C for more information.

Neurodevelopmental Disorders

Mental retardation is a neurodevelopmental disorder that, in some cases, is related to exposures to environmental contaminants such as lead. A measure of mental retardation in children is presented here. A second neurodevelopmental disorder, attention-deficit/hyperactivity disorder, is discussed in the Emerging Issues section of this report. Although the studies described above have related lead, PCBs, mercury, and perhaps other contaminants to adverse neurodevelopmental effects in humans, it is not currently possible to determine the extent to which environmental contaminants contribute to developmental disorders.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants: lead (E1-E2)	Lead in Blood (B1-B3)		
	Drinking Water: lead (E6-E7)	Mercury in Blood (B4)	*Neurodevelopmental (D7)*	Lead in Schools (S1-S3)

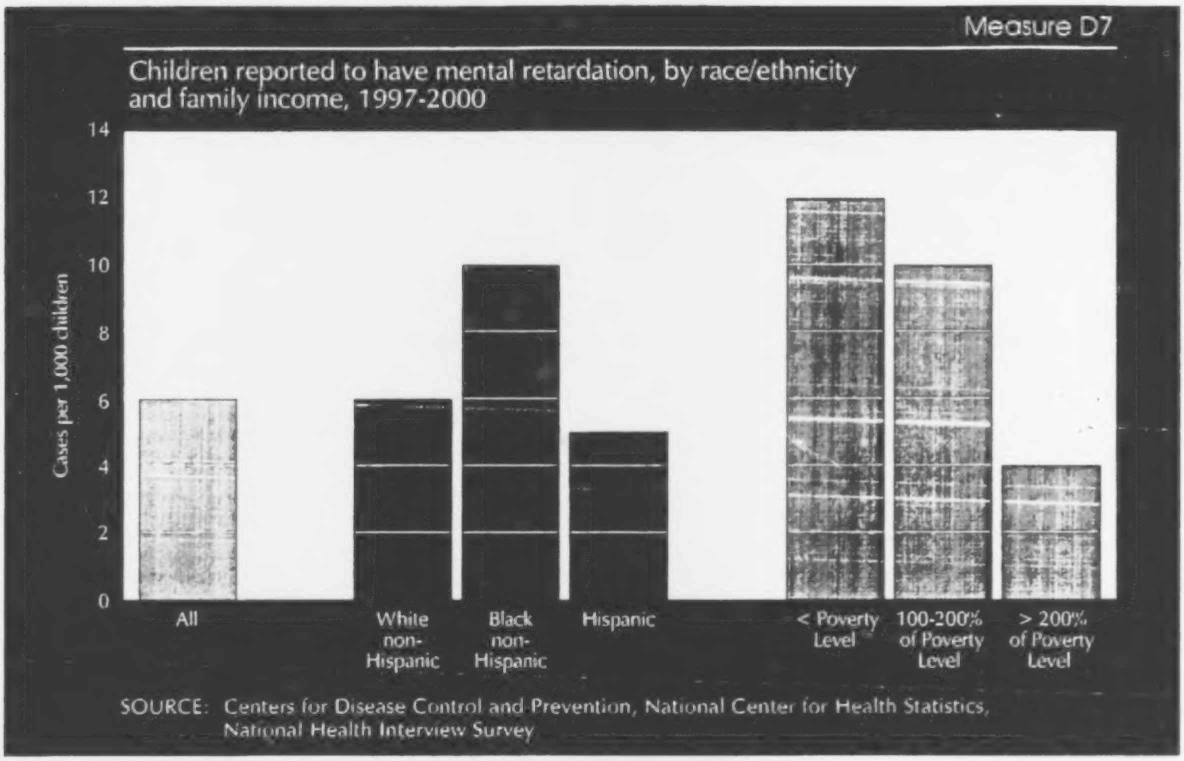
Mental Retardation

The most commonly used definitions of mental retardation emphasize subaverage intellectual functioning before the age of 18, usually defined as an intelligence quotient (IQ) less than 70 and impairments in life skills such as communication, self-care, home living, and social or interpersonal skills. Different severity categories, ranging from mild retardation to severe retardation, are defined on the basis of IQ scores.[82]

Researchers have identified many causes of mental retardation, including genetic disorders, traumatic accidents, and prenatal events such as maternal infection or exposure to alcohol.[82, 83] Exposure to lead and exposure to particularly high levels of mercury also have been shown to cause mental retardation.[66-67, 84] Furthermore, lead, mercury, and PCBs all have been found to have adverse effects on intelligence and cognitive functioning in children. Exposure to these environmental contaminants therefore has the potential to increase the proportion of the population with IQ less than 70, thus increasing the incidence of mental retardation in an exposed population.[85]

The causes of mental retardation are unknown in 30 to 50 percent of all cases.[83] The causes are more frequently identified for cases of severe retardation (IQ less than 50). The cause of mild retardation (IQ between 50 and 70) is unknown in more than 75 percent of cases.[86, 87]

This measure on the prevalence of mental retardation among U.S. children presents data obtained from the National Health Interview Survey (NHIS). Although the NHIS provides the best national-level data available, NHIS data likely underestimate the prevalence of mental retardation. Reasons for this understatement may include late identification of affected children and the exclusion of institutionalized children from the NHIS survey population. Further, the NHIS relies on parents reporting that their child has been diagnosed with mental retardation, and accuracy of parental responses could be affected by cultural and other factors.



Measure D7

Children reported to have mental retardation, by race/ethnicity and family income, 1997-2000

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

- In 1997-2000, about 6 children out of every 1,000 were reported to have been diagnosed with mental retardation.
- Reported rates of mental retardation were 10 per 1,000 Black non-Hispanic children, 6 per 1,000 White non-Hispanic children, and 5 per 1,000 Hispanic children.
- 12 children out of every 1,000 living in families with incomes below the poverty level were reported to have mental retardation. Reported rates of mental retardation were lowest for children living in families with higher incomes.

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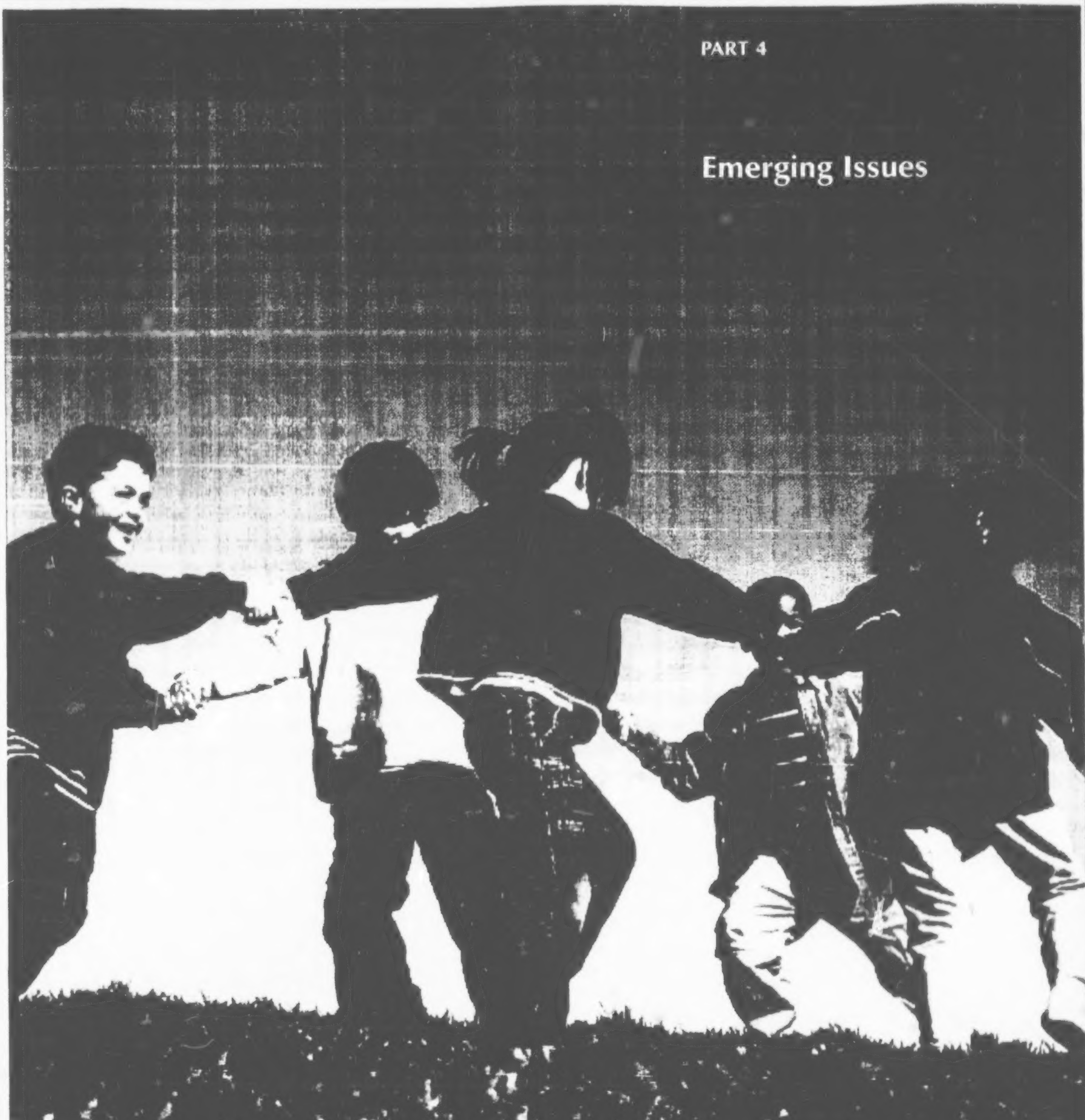
PART 4

Emerging Issues



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Emerging Issues

The links between environmental contaminants and childhood diseases and disorders are receiving increasing attention and research. We can expect that our understanding of how children's health may be influenced by environmental factors will continue to improve with more research and better data about both environmental contaminants and health outcomes.

This section presents information about important aspects of children's environmental health for which data recently have become available. Additional research in these areas will be useful to better determine how particular exposures to environmental contaminants might contribute to these particular health areas. It is important to identify emerging issues and new data sources in order to continue to expand our understanding of children's environmental health.

This section includes two emerging issues: mercury concentrations in fish, and attention-deficit/hyperactivity disorder (ADHD).

Mercury in Fish

Some pollutants build up in the bodies of fish and other animals, reaching high concentrations at upper levels of the food chain. For mercury, this "bioaccumulation" process occurs primarily in aquatic systems such as lakes or oceans. Mercury that is deposited from air currents or released to water bodies tends to become attached to particles and deposited into sediments. There, under certain conditions, bacteria convert metallic or elemental forms of mercury into methylmercury.[1] Methylmercury can be absorbed in particles or from the water by small creatures such as shrimp or other invertebrates, which then are consumed by predators including fish.[2] As each organism builds up methylmercury in its own tissues, and as smaller fish are eaten by larger fish, concentrations of methylmercury can accumulate, particularly in those of large fish that live a relatively long time.[3-6] Examples of other chemicals that bioaccumulate include dioxins, PCBs, and chlorinated pesticides such as DDT or chlordane. Some of these chemicals also may pose risks to children.

Fish are the most common source of exposure to methylmercury for most people in the United States[7, 8] and in many countries around the world.[9] As noted in Part 2 of this report, about 8 percent of women of child-bearing age have blood mercury concentrations greater than 5.8 parts per billion (equivalent to EPA's reference dose) based on data from the Centers for Disease Control and Prevention. EPA has determined that children born to such women may be at some increased risk of potential adverse health effects.[10] Chemicals accumulated by women may pass through the umbilical cord, contributing to prenatal exposure in children. Prenatal exposure to such levels of methylmercury may cause developmental and cognitive effects in children, even at doses that do not result in effects in women who are or may become pregnant.[11-13]

Some proportion of mercury that ends up in fish originates as emissions to the air. Mercury released into the atmosphere can travel long distances on global air currents and be deposited in areas far from its original source.[14, 15] The largest human-generated source of mercury emissions in the United States is the burning of coal, which is roughly one percent of mercury in the global pool. Other sources include the combustion of waste and industrial processes that use mercury.[14]

Information regarding warnings to the public about elevated concentrations of methylmercury in fish provides some indication of the likelihood of exposure to mercury from fish that people catch for their own use. Fish advisory information is not a surrogate for exposure to the general population, because most people eat only commercial fish that they purchase in stores or restaurants. However, there are subpopulations who do consume fish they have caught from waters covered by fish advisories, and fish advisory information is an indirect surrogate for exposure to these populations.

The scope of the warnings issued by states varies considerably. States typically advise people to reduce their consumption of contaminated fish by switching to less-contaminated species or to smaller fish that have not accumulated as much mercury. For methylmercury, which accumulates in muscle tissue, changes in cooking practices such as trimming fat or cooking over a grill do not reduce exposure. States often provide guidance about the maximum number of meals of fish that can be safely consumed. Some warnings apply to entire states, others are issued for individual lakes or streams. States also issue warnings for other contaminants besides mercury.

Emerging Issues

A review of fish advisories for mercury indicates the following:

- Most states issue advisories to warn people about elevated concentrations of mercury in non-commercial fish. In 2001, 44 states had advisories in effect for mercury in non-commercial fish. In some cases, advisories tell people to avoid eating fish from a particular area or a particular species. In other cases, they tell people to limit the amount of fish that they consume in general from a specified body of water. Some advisories are directed at protecting particularly susceptible groups, usually women of child-bearing age and children.
- Statewide advisories have shown the greatest increase. In 2001, statewide advisories were in effect for 17 states—Connecticut, Indiana, Kentucky, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, New Hampshire, New Jersey, North Carolina, North Dakota, Ohio, Pennsylvania, Vermont and Wisconsin—up from five states in 1995. Another nine states have statewide advisories for mercury in their coastal waters. Increased public health concerns have led to increased monitoring and this may explain, in part, the observed increase in statewide advisories.
- State programs for monitoring contaminants in fish and issuing advisories vary greatly. The absence of a state advisory does not necessarily indicate that there is no risk of exposure to unsafe levels of mercury in recreationally caught fish. Likewise, the presence of a state advisory does not indicate that there is a risk of exposure to unsafe levels of mercury in recreationally caught fish, unless people consume these fish at levels greater than those recommended by the fish advisory.
- Although some states monitor fish in a large number of water bodies, other states monitor few or none. The relationship between monitoring, setting of fishing advisories, and frequency of fishing has not been evaluated by EPA. Also, the concentration of mercury that triggers an advisory varies from state to state. As a result, the number of advisories does not directly represent the severity of contaminants in recreationally caught fish. It also reflects the extent of monitoring and the way that states assess risk.

EPA will work with other agencies to evaluate the feasibility of developing a measure more closely related to exposures that reflect mercury concentrations in fish intended for human consumption, for inclusion in future reports.

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
		Mercury in Blood (B4)	Neurodevelopmental (D7)	

Attention-Deficit/Hyperactivity Disorder

Attention-deficit/hyperactivity disorder (ADHD) is a disruptive behavior disorder characterized by ongoing inattention and/or hyperactivity-impulsivity occurring in several settings and more frequently and severely than is typical for individuals in the same stage of development.[17] ADHD can make family and peer relationships difficult, diminish academic performance, and reduce vocational development.

A diagnosis of ADHD considers whether a child is hyperactive, inattentive, or impulsive, at levels that are higher than expected for a child's developmental stage, and whether the behaviors occur on a continual basis in different settings (for example, both at school and at home) and whether the behaviors interfere with the child's ability to function in those settings.[17, 18]

As the medical profession has developed a greater understanding of ADHD through the years, the name of this condition has changed. The American Psychiatric Association adopted the name "attention deficit disorder" in the early 1980s and revised it to "attention-deficit/hyperactivity disorder" in 1987.[19]

Research on this disorder is ongoing and extensive, and new findings are frequently reported, but the causes of ADHD are unknown. Research indicates that there are genetic influences on the incidence of ADHD.[18, 20-22] The role of environmental contaminants in contributing to ADHD is unknown, as few studies have looked explicitly at the relationship between ADHD and exposures to environmental contaminants. However, many of the behaviors that are observed in children with ADHD also have been associated with elevated exposures to certain environmental contaminants. Several studies have found relationships between attention problems, hyperactivity, and impulsivity, which are the common behaviors of ADHD, and exposures to lead[23-28] and PCBs.[29-32] Animal studies provide supporting evidence that exposures to PCBs and lead may contribute to ADHD.[28, 33]

Data on the prevalence of ADHD among U.S. children are available from the National Health Interview Survey (NHIS), conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention, for the years 1997-2001. Although the NHIS provides the best data available, it is difficult to develop estimates of the prevalence of ADHD for a variety of reasons. Diagnosis of ADHD relies on recognition of various types of behaviors in different combinations, and therefore requires a certain amount of judgment on the part of a doctor, similar to other psychiatric disorders. Many other problems, including anxiety disorders, depression, and learning disabilities, can be expressed with signs and symptoms that resemble those of ADHD. As many as half of those with ADHD also have other mental disorders, which can make it harder to diagnose and treat ADHD.[34]

A diagnosis of ADHD depends not only on the presence of particular symptoms and behaviors in a child, but on concerns being raised by a parent or teacher about the child's behavior and on the child's access to a doctor to make the diagnosis. Further, the NHIS relies on parents reporting that their child has been diagnosed with ADHD.

It is unclear whether the percentage of children with ADHD has increased in recent years. Although recently more children have been diagnosed with and treated for ADHD, this increase may not reflect an increase in incidence, but rather greater awareness of the condition due to media attention, development of effective treatments, or other factors. Continued tracking of ADHD in the coming years should be useful for evaluating trends in diagnosis of ADHD.

Emerging Issues

It is most informative to focus on ADHD statistics for children 5-17 years old, because it is difficult to diagnose ADHD in younger children. Data from the NHIS indicate that:

- In 1997-2000, 6.7 percent of children ages 5-17 were reported to have been diagnosed with attention-deficit/hyperactivity disorder (ADHD).
- Eight percent of White non-Hispanic children, 5 percent of Black non-Hispanic children, and nearly 4 percent of Hispanic children were reported to have ADHD.
- Almost 14 percent of White non-Hispanic children living in families with incomes below poverty level were reported to have ADHD—the highest of any group.
- Two to three times more boys than girls are diagnosed with ADHD.[35]

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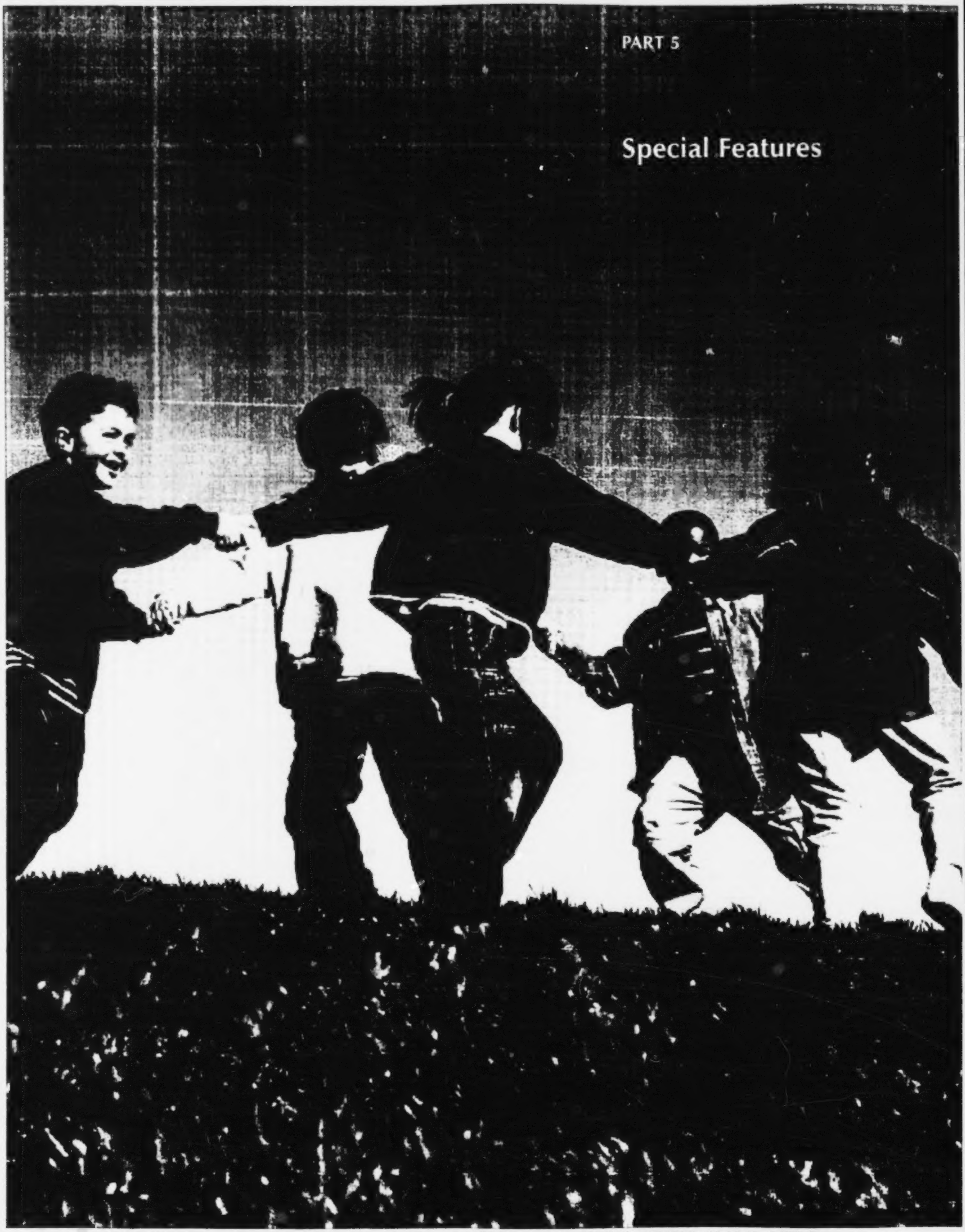
PART 5

Special Features



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Special Features

This Special Features section presents measures of environments and health disorders for which data are available only for individual states, not for the nation as a whole.

The measures in this section address potential exposures to lead and pesticides at schools, an environment where children spend a significant portion of their time. The data on lead in schools are from California; the data on pesticides in schools are from Minnesota. The final measure in this section presents trends in birth defects, a collection of childhood conditions for which there is some suspected environmental influence, using data from California.

Lead in California's Public Elementary Schools

Elevated levels of lead in blood remain an important childhood environmental health hazard in the United States. Childhood exposure to lead contributes to learning problems such as reduced intelligence and cognitive development.[1-3] Studies also have found that childhood exposure to lead contributes to attention-deficit/hyperactivity disorder[4] and hyperactivity and distractibility;[5-7] increases the likelihood of dropping out of high school, having a reading disability, lower vocabulary, and lower class standing in high school;[8] and increases the risk for antisocial and delinquent behavior.[9] There is no demonstrated safe concentration of lead in blood, and adverse health effects can occur at very low blood lead levels.[2, 3, 10]

Ingestion of dust and soil contaminated mainly by deteriorated lead-based paint and by past emissions of leaded gasoline deposited in the soil are the main sources of lead exposure.[11-13] Direct ingestion of paint chips can be important in some cases.[14] Other sources of lead exposure in the United States include drinking water, soil and dust, canned food and drink, lead-glazed ceramics, and industrial plant emissions.[11, 15]

In 1992, the California Legislature approved the Lead-Safe Schools Protection Act. Following approval of this legislation, the California Department of Health Services conducted a study to determine the prevalence of lead and lead hazards in the state's public elementary schools, including elementary school buildings that house day care centers and preschools. Measures S1-S3 present data from this hazard assessment.

Healthy People 2010:	Objective 8-11 of Healthy People 2010 aims to eliminate elevated blood lead levels in U.S. children. See Appendix C for more information.

Deteriorated Lead-containing Paint in California's Public Elementary Schools

The presence of lead-containing paint in environments where children reside or spend time—including homes, schools, and childcare facilities—does not always result in exposure. However, it creates a potential for exposure, particularly if the paint is deteriorated and accessible to children.

Deteriorated paint is more prevalent in old and poorly maintained buildings. According to the National Center for Education Statistics, 75 percent of all public elementary schools nationwide were built before 1970, and about 14 percent of these schools have never been renovated.[16] Sixteen percent of these pre-1970 schools were renovated before 1980, the period preceding and immediately after the Consumer Product Safety Commission's 1978 rule limiting the amount of lead allowed in paint to 600 parts per million.[16] Therefore, about one-third of the nation's oldest schools (those built before 1970) were never renovated or were renovated before limits were placed on the amount of lead in paint. Recent housing survey estimates from the U.S. Department of Housing and Urban Development (HUD) suggest that 25 percent of housing units with one or more children under the age of 6 have significant lead-based paint hazards.* [17, 18]

Children under the age of six are particularly vulnerable to lead exposure and its adverse effects because of age-related risk factors such as hand-to-mouth behavior, pica (a tendency to mouth or attempt to consume non-food objects),[19] small body mass, and a developing brain and nervous system. Currently, 60 percent of children aged three to five are enrolled in day care or similar programs and more than 7.8 million children are enrolled in pre-kindergarten to first grade.[20] Lead hazards within the school environment may be an important contributor to exposure.

In its study of the prevalence of lead and lead hazards in California public elementary schools, the California Department of Health Services collected a maximum of four interior and seven exterior paint chip samples from each of the 200 schools studied. Where possible, paint chip samples were obtained from areas where the paint was visibly deteriorated.

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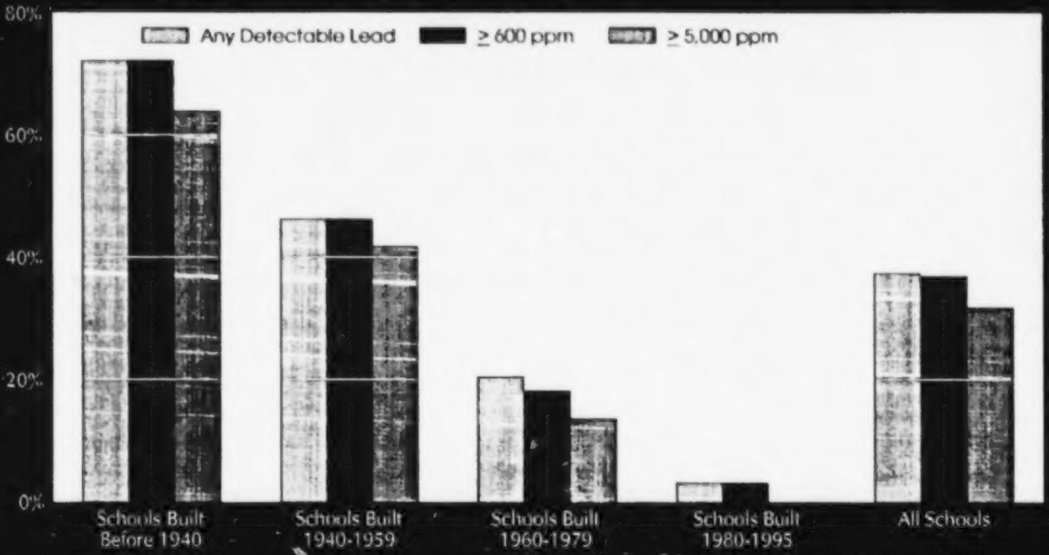
* The Department of Housing and Urban Development defines a "significant lead-based paint hazard" as: a) deteriorated lead-based paint (paint containing 0.5 percent by weight or 1 milligram per square centimeter of lead) of more than 20 square feet (exterior) or two square feet (interior) on large-surface-area components, or damage to more than 10 percent of the total surface area of interior small surface area components; or b) lead-contaminated dust on floors with 40 micrograms or greater of lead per square foot, dust on window sills with 250 micrograms or greater of lead per square foot; or c) bare lead-contaminated soil of more than 9 square feet with a soil lead concentration of 1,200 parts per million or greater, or 400 parts per million for bare soil in an area frequented by a child under the age of six.

Measure S1

Percentage of California public elementary schools with lead paint and some deterioration of paint, 1994-1997



SOURCE: California Department of Health Services, Childhood Lead Poisoning Prevention Branch. *Lead Hazards in California's Public Elementary Schools and Child Care Facilities.* April, 1998

- Thirty-seven percent of all public elementary schools surveyed in California had both lead-containing paint and some deterioration of paint. Thirty-two percent of these schools had lead-based paint and some deterioration. The term "lead-containing paint" refers to paint containing any detectable level of lead. "Lead-based" paint refers to paint containing at least 5,000 parts per million of lead.
- Generally, the proportion of schools with lead-containing paint and some deterioration of paint decreased as the age of the schools decreased. Most (72 percent) of the California schools built before 1940 had lead-containing paint and some deterioration, compared with only 3 percent of the schools built between 1980 and 1995. A similar trend was observed for paint deterioration and lead in paint at or exceeding the Consumer Product Safety Commission and EPA/HUD standards (600 parts per million and 5,000 parts per million respectively).
- Ninety percent of all schools surveyed had lead-containing paint. All pre-1980 schools and 45 percent of schools built between 1980 and 1995 had lead-containing paint. (Data not shown.)

Related Measures:	**Environmental Contaminants**	**Body Burdens**	**Childhood Illnesses**	**Special Features**
	Criteria Air Pollutants: lead (E1-E2) Drinking Water: lead (E6-E7)	Lead in Blood (B1-B3)	Neurodevelopmental (D7)	*Lead in Schools (S1-S3)*

Lead-contaminated Soil Near California's Public Elementary Schools

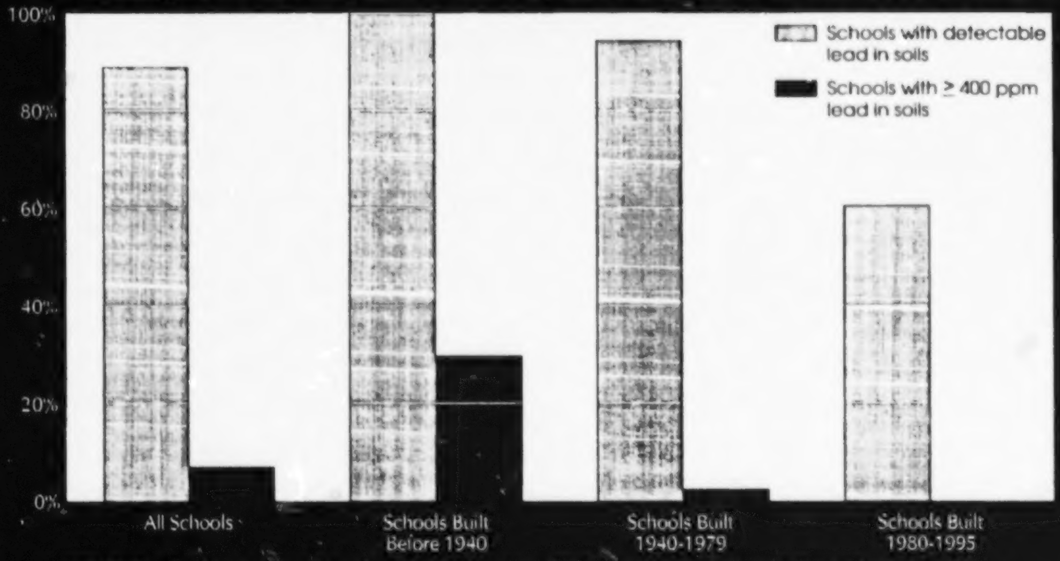
Lead-contaminated soil poses an exposure risk to children. EPA's hazard standard for lead in soil is 400 parts per million by weight in play areas, and 1,200 parts per million in bare soil in the remainder of the yard. EPA recommends measures to reduce exposure when lead in soil is at or above these hazard standards.[21] Exposure to soil lead levels lower than the hazard standards also may pose some risk. Current research shows there is no safe level of lead in blood.[2, 3, 10]

Deteriorated exterior lead-based paint and fallout from air-borne emissions may lead to contamination of soil in schoolyard areas. Some of the widespread lead in U.S. soils, especially around busy roadways, is attributable to automobile emissions before leaded gasoline was phased out.[22, 23]

Lead in California Schools

Measure S2

Percentage of California public elementary schools with lead in soils, 1994-1997



SOURCE: California Department of Health Services, Childhood Lead Poisoning Prevention Branch. *Lead Hazards in California's Public Elementary Schools and Child Care Facilities.* April, 1998

- Eighty-nine percent of all California schools in the study (public elementary schools) had detectable levels of lead in soils. Only 7 percent of the schools had lead levels in soil at or exceeding the EPA hazard standard.
- All buildings built before 1940 had detectable levels of lead in soils, and 30 percent exceeded the EPA hazard standard.
- None of the schools built after 1980 had levels of lead in soils at or exceeding the EPA hazard standard.
- The typical lowest concentration of lead that the method could measure for the soil sample analysis was 20 parts per million, with a range of 3.7 parts per million to 151 parts per million.

Related Measures:	Environmental Contaminants	Body Burdens	Childhood Illnesses	Special Features
	Criteria Air Pollutants: lead (E1-E2) Drinking Water: lead (E6-E7)	Lead in Blood (B1-B3)	Neurodevelopmental (D7)	*Lead in Schools (S1-S3)*

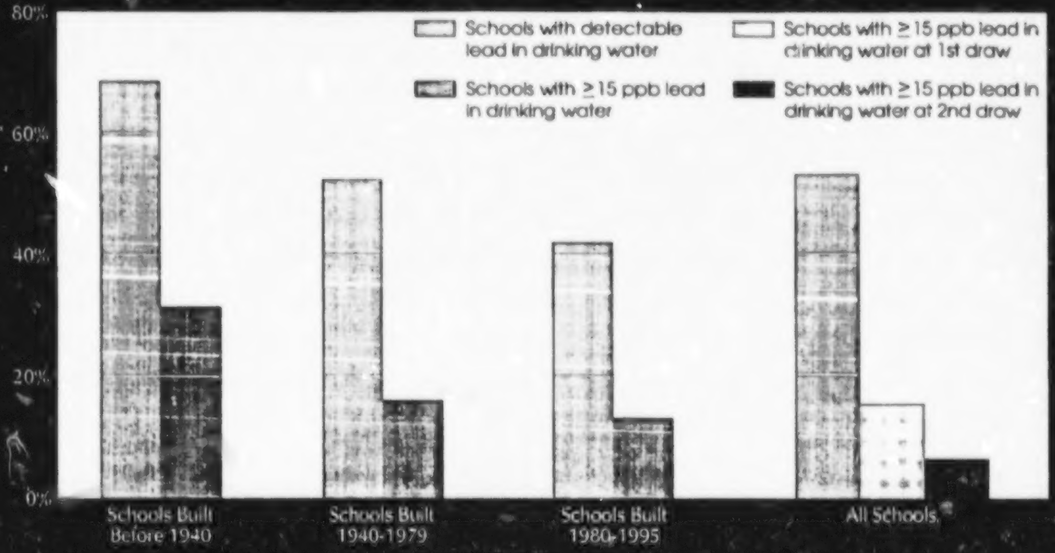
Lead-contaminated Drinking Water in California's Public Elementary Schools

Drinking water may be contaminated with lead through contact with lead pipes found in older buildings, brass faucets, and copper pipes with lead solder. The use of pipes, plumbing fittings, fixtures, or flux that are not "lead-free" in the installation and repair of non-residential facilities was prohibited in June, 1986.[24] EPA's action level for lead in drinking water is 15 parts per billion, and the Maximum Contaminant Level Goal (MCLG) is zero.[25] The MCLG is the concentration in drinking water at which no known or anticipated adverse effect on the health of persons would occur and which allows an adequate margin of safety.

On average, lead in drinking water accounts for an estimated 10-20 percent of total lead exposure in young children.[26] "First draw" water from pipes that have not been flushed tends to have higher lead content but are not representative of typical concentrations.[26]

Measure S3

Percentage of California public elementary schools with lead in drinking water, 1994-1997



SOURCE: California Department of Health Services, Childhood Lead Poisoning Prevention Branch. *Lead Hazards in California's Public Elementary Schools and Child Care Facilities*. April, 1998

- Detectable amounts of lead were reported in drinking water at 53 percent of all schools in the California study. First draw samples from 15.5 percent of participating schools (31 out of 200 schools) exceeded the EPA hazard standard of 15 parts per billion. Drinking water from approximately 6.5 percent of participating schools remained above the standard on the second draw. Second-draw samples are more representative of the lead concentrations that children are exposed to during most of the day. Data for first- and second-draw samples are available only for all schools combined, and not for schools grouped by year of construction.
- The percentage of schools with lead contamination exceeding the EPA standard decreased as the age of the schools decreased.
- The lowest concentration of lead measurable in the water analysis was 5 parts per billion.

Related Measures:	Environmental Contaminants	Body Burdens	Childhood Illnesses	Special Features
	Criteria Air Pollutants: lead (E1-E2) Drinking Water: lead (E6-E7)	Lead in Blood (B1-B3)	Neurodevelopmental (D7)	*Lead in Schools (S1-S3)*

Pesticide Use in Minnesota's Public and Private Schools

Legislation recommending the use of Integrated Pest Management (IPM) in schools was introduced in Minnesota in 1999 and again in 2000. In response to increased awareness and concern about the issue of pesticides in schools, the state conducted a survey in 1999 on current pest management practices in kindergarten through secondary (K-12) schools.

This measure presents data from the survey on the frequency of pesticide use and some information about how these pesticides were used. The measure is only a surrogate for exposure. The frequency with which pesticides are used is an indicator of potential exposure because the risk of exposure increases with the frequency of pesticide use. This measure does not provide information about the toxicity of pesticides used or details about how they were applied and thus cannot provide a complete representation of the risk of adverse effects following exposure.

Pesticides in Minnesota Schools

Pesticide Use in Schools

Children may be exposed to pesticides in their diets and through contact with pesticides used in homes, schools, and day care centers. Children are particularly vulnerable to the effects of pesticides because of their unique susceptibilities and relatively high exposures compared with adults.[27-30] Because children spend a significant portion of time at school, this setting may be an important contributor to overall exposure.

There is no federal statute requiring the collection of data on pesticide use in schools; thus there is no nationwide information on the amount of pesticides used in the nation's 110,000 schools.[31]

Few states require reporting of pesticide use. For those states that do, the information collected generally is not adequate to assess exposure. Some states have regulated and/or assessed the use of pesticides in schools. In 1995, Louisiana passed a law requiring its school districts to report the amount of pesticides used annually. In New York, commercial applicators are required by a 1996 law to report the amount of a specific pesticide used and the location where it was applied. Six states—Arizona, California, Connecticut, Massachusetts, New Hampshire, and New Mexico—require commercial applicators to report the amount of specific pesticides used, but not the locations where the pesticides are applied.[31]

In recent years, EPA has recommended that schools undertake Integrated Pest Management (IPM) to reduce pesticide use. An IPM program for schools may include redesigning and repairing structures, improving sanitation, employing pest-resistant varieties of plants, establishing watering and mowing practices that minimize the need for pesticides, and applying pesticides judiciously. IPM programs frequently are more economical and less hazardous to people, the environment, and property than conventional approaches to pest control.[32] Currently, approximately 10 states require the adoption of IPM in schools.[33]

In 1999 the state of Minnesota conducted a survey on pesticide use in schools. The results presented here focus on indoor uses of pesticides, because pesticide residues can be persistent indoors,[34-36] and because children spend most of their time indoors when they are at school. Some pesticides have been detected at indoor concentrations potentially hazardous to children weeks and months after application.[34, 37]

Healthy People 2010: Objective 8-24 of Healthy People 2010 aims to reduce exposure to pesticides. See Appendix C for more information.

Birth Defects

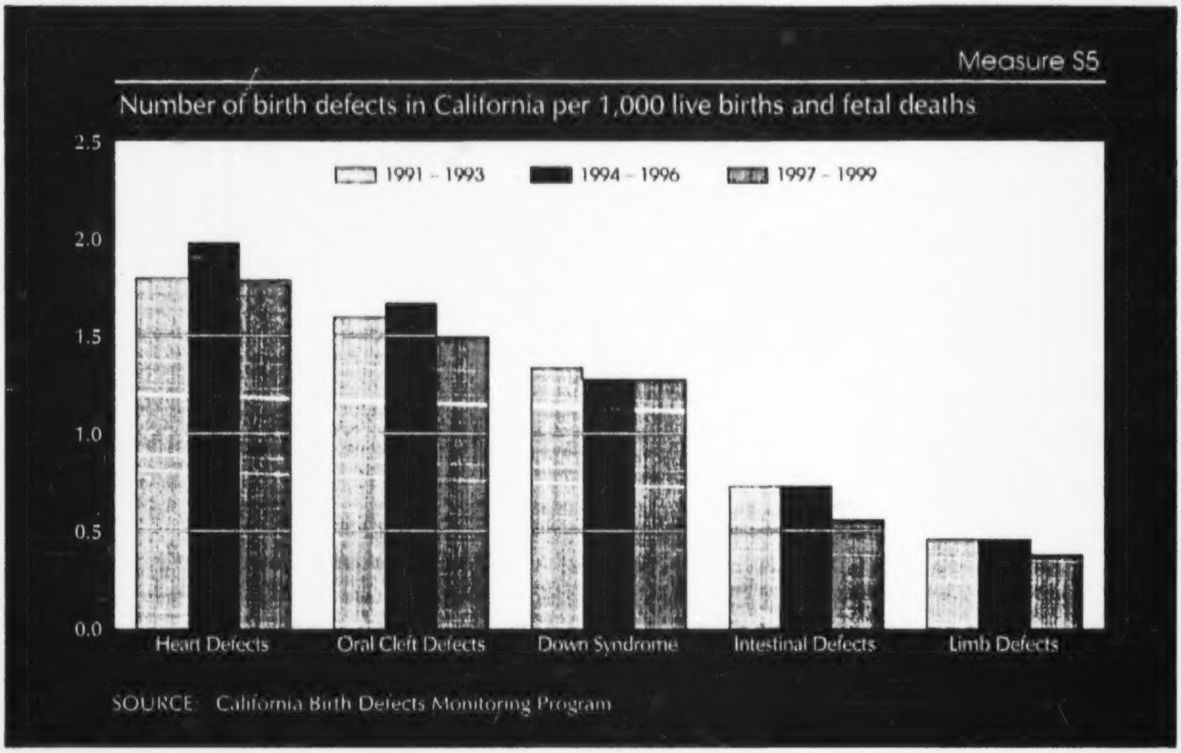
Birth defects are the leading cause of infant death in the first year of life, accounting for about 20 percent of infant deaths in 1999.[39] The term "birth defects" covers a range of structural defects of the limbs or mouth, defects that affect development of the spinal cord, and defects of internal organs, such as the heart. Infants who do not die from birth defects often have lifelong disabilities, such as mental retardation, heart problems, or difficulty in performing everyday activities such as walking.

Some birth defects are inherited. Other risk factors for birth defects include prenatal exposure of the fetus to certain pharmaceuticals, such as Accutane; alcohol; and insufficient folate in a woman's diet. The causes of a significant portion of birth defects are unknown, but research suggests that defects could be influenced by environmental factors.[40] Several environmental contaminants cause birth defects when pregnant women are exposed to high concentrations. Mercury poisoning in Minamata, Japan, resulted in birth defects such as deafness and blindness.[41] Prenatal exposures to high concentrations of polychlorinated biphenyls (PCBs) have resulted in stained and acned skin and deformed nails in children.[42] However, the relationship between exposure to lower concentrations of environmental contaminants and birth defects is less clear.

A number of epidemiological studies have evaluated the relationship between environmental and occupational exposures and birth defects. A recent scientific review that evaluated multiple studies of women's occupational exposure to organic solvents found an increased risk for birth defects such as heart defects and oral cleft defects.[43] Studies of fathers have found that certain occupations are associated with birth defects in their children.[44-46] Studies evaluating the role of pesticides in birth defects have found an association between maternal and paternal exposure to pesticides and increased risk of offspring having or dying from birth defects.[44-54]

There currently is no national monitoring system for birth defects. However, most states have some type of birth defects monitoring program. At the end of 2000, 45 of the states, the District of Columbia, and Puerto Rico had some type of existing birth defects monitoring program.[55] The type of tracking varies widely among the states. A small portion of these states have the most complete type of tracking system, which includes actively researching medical records for birth defects and following children through the first year of life. The remaining states have some type of monitoring program, but do not have all the aspects of a complete surveillance system. California has monitored birth defects since 1983 and has a monitoring program that is considered most complete. Data from California for several major defects are presented here.

Birth Defects in California



Measure S5

Number of birth defects in California per 1,000 live births and fetal deaths

SOURCE: California Birth Defects Monitoring Program

- Heart defects are the most common birth defect in California, with 1.8 cases per 1,000 live births and fetal deaths in 1997-99.
- The rates of birth defects in California generally remained constant during the 1990s.
- Other important defects not shown here are neural tube defects and defects of the reproductive system, such as hypospadias. During the 1990s, there were six cases of neural tube defects per 10,000 (0.6 per 1,000) live births and fetal deaths. There were insufficient data to determine a trend over the 1990s. Data on hypospadias are not available from the state of California.

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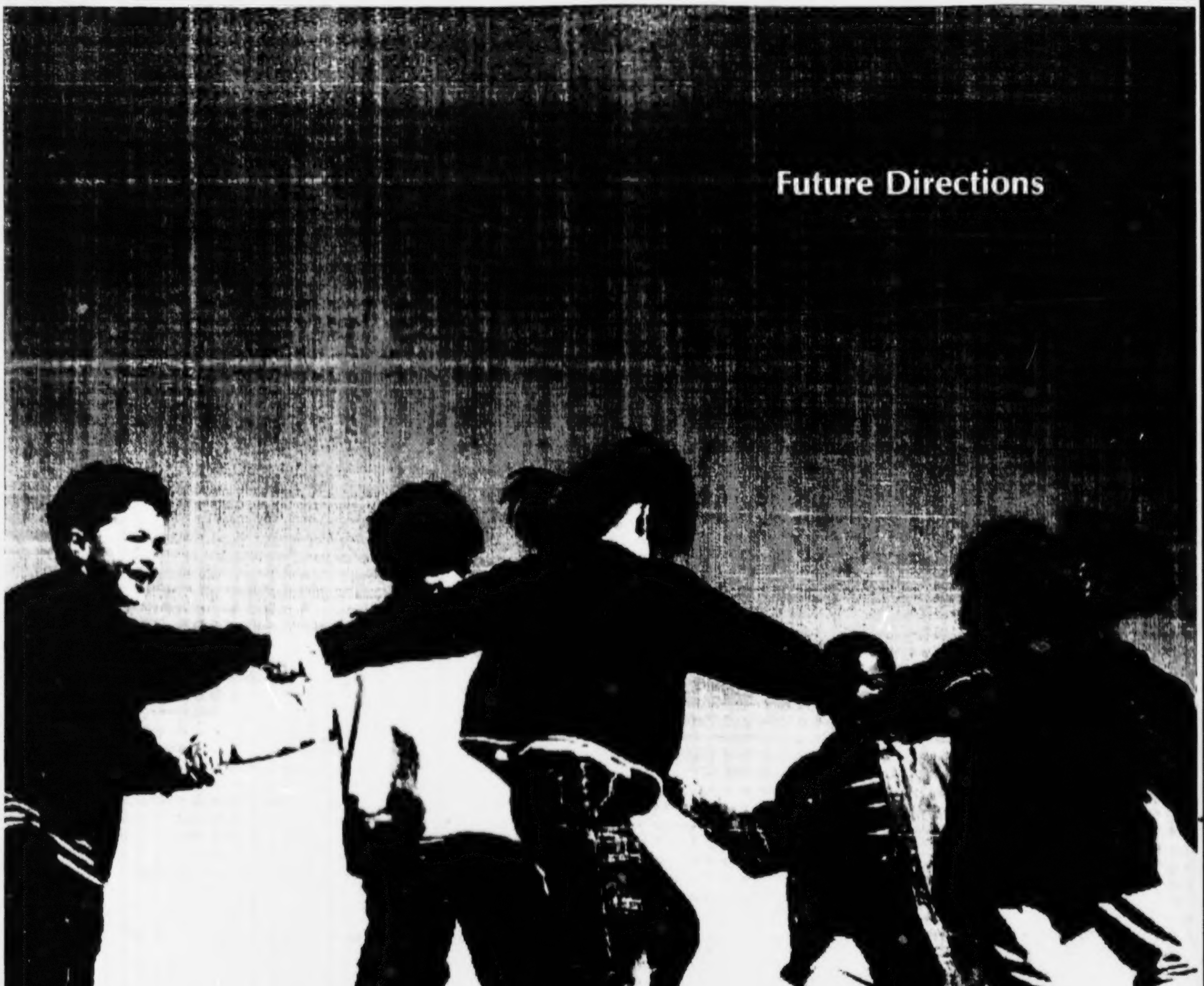
Future Directions



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Future Directions for *America's Children and the Environment*

This section discusses improvements that could be made to the measures in the report, improvements to the data sources used for the measures, and new measures that may be included in future reports.

Characteristics of Ideal Measures

Ideally, data sources for measures in all three parts of the report would provide information collected in a consistent manner for all of the nation's children. Data also would be available for 10 years or more to provide information about changes over time, and to show whether the changes were statistically significant. Information would be available on differences among geographic areas, by race/ethnicity, and by economic status.

For environmental contaminants, ideal measures would be nationally representative measurements of concentrations of environmental contaminants in air, water, food, and soil that can affect children's health. The measures would reflect the potential for children to be exposed to these pollutants.

For concentrations of contaminants measured in children and women of child-bearing age, ideal measures would reflect concentrations of the key pollutants in their bodies that pose a risk of adverse health effects.

For childhood illnesses, ideal measures would identify the percentage of children in whom important health conditions may have been caused by or exacerbated in part by environmental contaminants. In addition, the data would permit characterization of subgroups of each disease for which environmental contributions to the conditions are most relevant.

Part 1: Environmental Contaminants

Common Air Pollutants

The measures for criteria air pollutants are based on three kinds of data: exceedances of national standards, reports of daily air quality generated through the Air Quality Index, and measured concentrations of air pollutants in all counties.

An important future direction would be to estimate health risks associated with ambient concentrations of criteria air pollutants. Such measures would link air quality data with health outcomes. This measure would be risk-based and would incorporate current knowledge about the link between air pollution and health outcomes.

Hazardous Air Pollutants

The measure in this report for hazardous air pollutants (air toxics) is based on data from the year 1996 only. The previous edition of the report showed data based on EPA's 1990 modeling of ambient concentration of hazardous air pollutants, but the 1990 results are not included in the current measure because of differences in the way the modeled results are reported for 1990 and 1996. Hazardous air pollutant data will be developed for every three years starting with 1996, and years after 1996 will be comparable. Future reports will incorporate trends in hazardous air pollutants and their possible relationships to children's health.

The measure in this report uses health benchmarks that are based on lifetime exposures, and the data and methods used to establish the benchmarks are based on responses in mature organisms (animals and humans). For future reports EPA will explore the availability of health benchmarks that are specifically based on effects from exposures during childhood.

Data from air toxics monitoring programs also could be considered for inclusion in future editions of this report. Currently, national data on air toxics monitoring are limited, and much of the monitoring and data collection are performed at the state level.

The hazardous air pollutant measures in this report also are limited in that they represent the presence of these pollutants in ambient air only. For certain hazardous air pollutants that are persistent in the environment, greater exposures occur in food. These pollutants settle out of the air onto land and into bodies of water, and are taken up in the food chain. This pathway of exposure is addressed in part by the current measure on mercury in fish. Related measures for other contaminants will be considered for future reports (see the section on food contaminants below).

Indoor Air Pollutants

Indoor exposure to secondhand smoke is represented by a surrogate measure reflecting the percentage of homes of children where people smoke. The most important improvement would be to add data about sources of other indoor air pollutants, such as consumer products, gas stoves, and furnishings, for both homes and schools. To date no nationally representative data on air contaminants in homes, schools, and other indoor environments in which children may spend large amounts of time have been identified, but efforts to explore possible measures will continue.

Drinking Water Contaminants

The measures for contaminants in drinking water reflect violations of national standards. These measures do not distinguish among the impacts of various concentrations of contaminants. The data on drinking water contaminants are less complete than those used for the air measures because there is less monitoring and reporting of water contaminants. In addition, the drinking water contaminant measures in this report rely on the Maximum Contaminant Level (MCL) standards. The MCL standards are based partly on health considerations but also take into account technical feasibility and cost-benefit considerations.

Each MCL also has a corresponding Maximum Contaminant Level Goal (MCLG), which is based only on health considerations. The MCLGs could be considered for measures in future reports.

Actual measured contaminant concentrations would provide the most relevant measures of potential risks to children. The most complete data on contaminants in drinking water are collected at the state level; information from the states would have to be compiled nationally to improve the measures for drinking water.

Another limitation of the data on drinking water is that many water systems do not adequately monitor for contaminants, so no information about potential risks to children in those areas is available. Future reports will consider data on such water systems collected at the state level.

Surface Water Contaminants

In the future, EPA would like to characterize the risks posed to children when they swim in waters contaminated with bacteria. Children are at greater risk of illness while swimming than adults are because of their longer exposure times and more frequent accidental ingestion of water. Data for monitoring recreational waters currently are being collected and will be considered for future reports.

Food Contaminants

Dietary exposures to pesticides are represented in this report by a measure of organophosphate pesticide residues on foods. This measure does not represent actual exposures or risks to children from pesticides.

As required by the Food Quality Protection Act, EPA currently is conducting a cumulative risk assessment for the organophosphate pesticides. For the first time ever, this scientific assessment evaluates the potential risks to children from the combined estimates of all contributing organophosphate residues in food and drinking water consumption, and from activities around the home. EPA already has imposed various restrictions on many individual uses of organophosphates, particularly those that may pose greater risk to children from dietary and residential sources. These restrictions, and others that may be imposed as a result of the cumulative assessment, are expected to lower children's potential exposure to these pesticides and thereby reduce potential health risks. EPA will evaluate the outputs from the cumulative risk assessments to determine how they may be used in developing measures that better reflect increases or decreases in pesticide exposure or risk. In addition, the Agency expects to add measures of pesticide exposures to the body burdens section of the report.

EPA also will examine the available data on the presence of other types of contaminants in foods. As noted above, some hazardous air pollutants find their way into the food chain after being deposited from the atmosphere, and their presence in food can pose more of a risk to women of child-bearing age than their presence in the air. This report includes information on states with fish consumption advisories for mercury. If feasible, future reports will better characterize the risks posed by the consumption of fish contaminated with mercury, PCBs, and other toxicants that affect neurological development, by including measures more closely related to exposures.

Finally, some children may be exposed to particularly elevated levels of contaminants in food, including children in homes where much of the diet comes from subsistence fishing. EPA will explore the availability of suitable data regarding such differential exposures for future reports.

Land Contaminants

For contaminants in soil, this report includes a measure of the percentage of children living within a mile of a Superfund site. This measure has the advantage of assessing only children who live near a site, but living near a site does not necessarily mean that they are exposed to contaminants. Also, there are other types of sites and land that may be contaminated but are not classified as Superfund sites. The most important improvement to this measure would be to include data on contaminants in soil, but nationally representative data are not currently available. A measure of children living in proximity to "Brownfield" sites also will be considered for future reports. State databases with information about contaminated sites also may be useful.

Future Directions

Other Contaminated Media

Key additional data needs for environmental contaminants focus on exposure pathways and environments that are particularly important for children. A number of contaminants may gather on household surfaces, including those found in indoor air, contaminants in soil that are tracked into the home, and those from the workplace that inadvertently are brought into the home on the parents' clothes or body. Young children may frequently be exposed to environmental contaminants that gather on floors and other surfaces in the home through hand-to-mouth and object-to-mouth contact. Data available for these exposure scenarios are limited.

Deteriorated lead-based paint, and the contaminated dust and soil it generates, is an important contributor to childhood lead exposures, particularly in the home. The U.S. Department of Housing and Urban Development (HUD) and the National Institute of Environmental Health Sciences recently conducted a survey of lead hazards in the home, with data collected from 1998-2000; HUD will repeat this survey in 2004. Measures drawn from the data gathered in these surveys will be considered for future editions of *America's Children and the Environment.*

Focusing on Children's Environments

This report includes measures related to potential exposures to children at school. Future work will consider other measures reflecting settings where children spend time. Additional measures related to where children live, go to school, and spend significant time (such as playgrounds) will be considered, as will measures on children's proximity to sources of exposures that may affect their health, such as highways, areas of high traffic, or hazardous waste sites.

Child care centers are a particularly important environment where many young children spend a substantial portion of their time. The U.S. Department of Housing and Urban Development, the Consumer Product Safety Commission, and EPA are conducting a survey of environmental hazards in child care centers. Data from this survey will be considered for future editions of this report, and the availability of other data on potential exposures to environmental contaminants in child care centers will be examined.

Part 2: Body Burdens

This report includes body burden data for lead and cotinine concentrations in the blood of children, and mercury concentrations in the blood of women of child-bearing age. These measures were chosen because it was possible to identify the health significance of the measured concentrations or multiple years of data were available. Future work will include more contaminants as multiple years of data become available. The development of methods for evaluating health implications of the available body burden data will be considered.

In future reports, EPA will present available body burden data (for women of child-bearing age) on contaminants for which prenatal exposure has been associated with childhood health effects, as with mercury. Cotinine will be considered as a possibility for this type of measure. In addition, future work will consider data related to contaminants in breast milk, which is an important source of exposure for infants.

Future Directions

Part 3: Childhood Illnesses

The current report includes measures for respiratory-related diseases, with an emphasis on asthma; measures for childhood cancer; and measures for neurodevelopmental disorders in children. All of these are complex conditions with multiple causes, and at present available data do not allow scientists to determine the role that environmental contaminants play in the prevalence of some childhood diseases.

Additional data collected by states or collected at the state level for some of the childhood illnesses will be assessed for future reports. In particular, future work will assess available data on childhood cancers by state.

In future work other datasets will be considered to supplement the estimates for neurodevelopmental disorders, which are based on parents' responses to the National Health Interview Survey. Potential data sources include registries of clinically diagnosed cases, which are available in select areas, and the percentage of children taking medications to treat certain neurodevelopmental disorders such as ADHD.

Additional measures reflecting emergency room visits for respiratory effects and hospitalizations for respiratory effects are included in this edition of the report. Minor respiratory symptoms, such as increasing cough and declines in lung function, are influenced by environmental factors but are not included in this report. Future work will focus on identifying appropriate data sources for such measures.

No nationally representative data are available for a number of other childhood diseases that may be in part caused by exposure to environmental contaminants such as birth defects and waterborne diseases. This report's Special Features section includes data on birth defects in California. Future reports will consider data from other states.

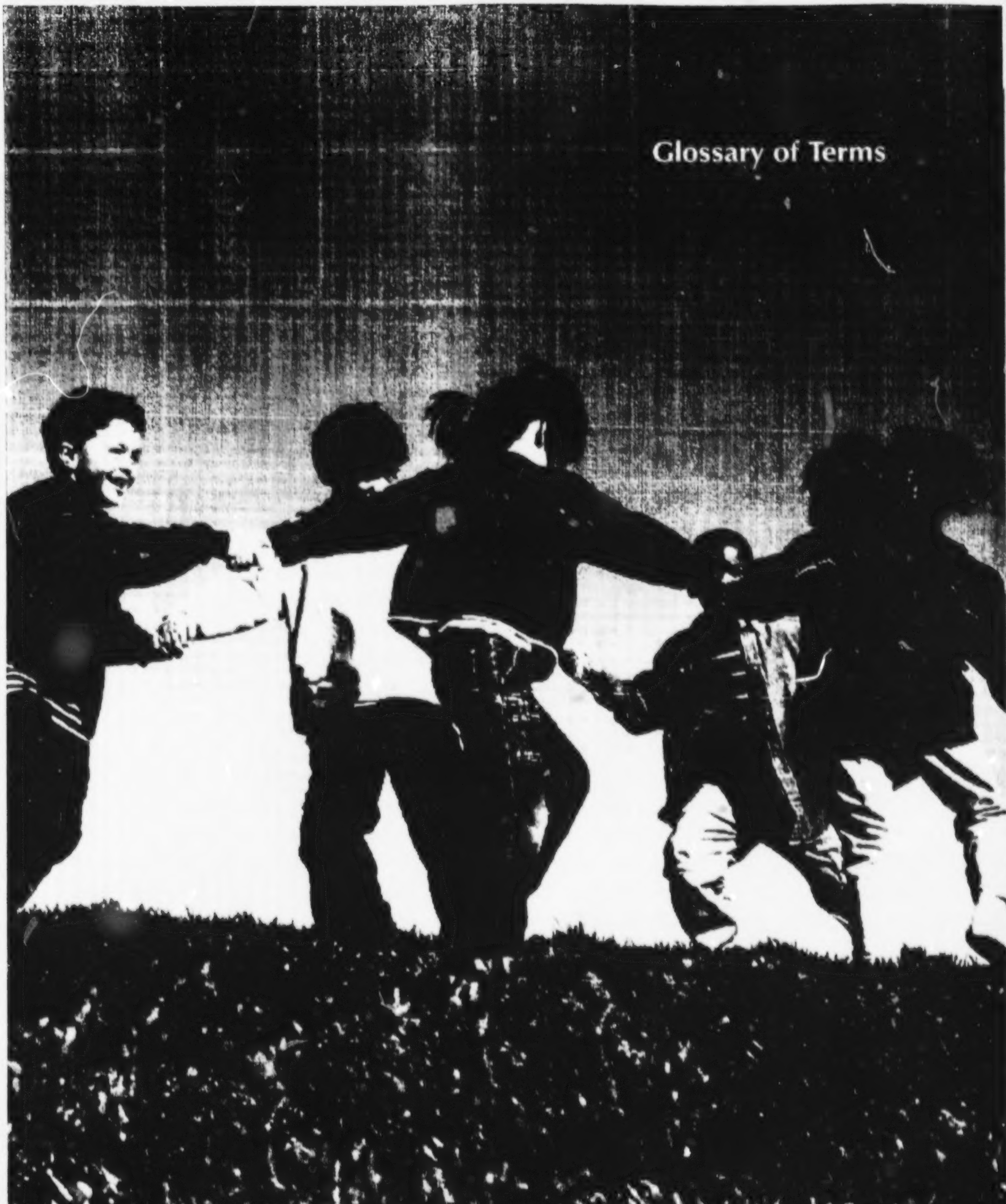
Environmental factors also may affect human reproduction, contributing to effects such as earlier age at puberty. These effects are important to monitor, and if suitable data become available they will be included in future reports. However, appropriate data sources for these and other important childhood diseases and disorders may not exist.

Part 5: Special Features

This report includes measures based on data from selected states on potential exposures to lead and pesticides in schools, and on birth defects. Future reports will include additional topics in this section that are important to children's health, but for which nationally representative data are not currently available. EPA will consider topics describing the contributions of different pathways of exposures to children, such as information on the multiple ways in which children may be exposed to pesticides.

In the future, EPA will evaluate new information on the sources of the contaminants in the environment now included in Part 1. This information would complete the path from source to contaminants to body burdens to health effects, and would help identify priorities for future policies.

Glossary of Terms



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Glossary of Terms

Air Toxics:
Synonym for "hazardous air pollutants." (See below).

Ambient Air:
Outdoor air, any unconfined portion of the atmosphere, open air.

Asthma:
A chronic inflammatory disorder of the lungs. Symptoms include wheezing, breathlessness, chest tightening, and cough.

Attention-Deficit/Hyperactivity Disorder (ADHD):
A disorder in which the prominent symptoms are hyperactivity, inattention, and impulsivity. Also referred to as ADD (attention deficit disorder).

Benzene:
A colorless, volatile, flammable, toxic liquid aromatic hydrocarbon (C_6H_6) used in organic synthesis, as a solvent, and as a component of motor fuel. Benzene is a known human carcinogen and an important hazardous air pollutant.

Cadmium:
A heavy metal used primarily for metal plating and coating operations, in applications such as transportation equipment, machinery and baking enamels, photography, and television phosphors. It also is used in nickel-cadmium and solar batteries, and in pigments. It also is found in cigarette smoke and is an important hazardous air pollutant.

Carcinoma:
A form of cancer that begins in the tissues lining or covering an organ.

Carbon Monoxide (CO):
A colorless, odorless, poisonous gas produced by incomplete combustion of fossil fuels; one of the six "criteria" pollutants for which EPA has set National Ambient Air Quality Standards under the Clean Air Act.

Carbon Tetrachloride:
A manufactured compound, most often found as a colorless gas. Because of its harmful effect on the ozone layer, the production and use of carbon tetrachloride in industrialized nations was banned in 1996 under the Montreal Protocol on Substances that Deplete the Ozone Layer. It is highly persistent and remains at levels of concern in the environment in the United States; it is an important hazardous air pollutant.

Cardiopulmonary Mortality:
Death due to malfunction of the heart and lungs; also refers to the death rate from these causes.

Cardiovascular Effects:
Health effects related to the heart and circulatory system.

Chlorinated Dibenzofurans (CDFs):
A family of 135 individual compounds with varying harmful health and environmental effects. CDFs typically are released to the environment through the incineration of municipal and industrial waste, accidental combustion of polychlorinated biphenyls (PCBs), and the manufacture of certain metals and paper products.

Chromium:
A heavy metal that is an important hazardous air pollutant. (See "heavy metals.") It is used for making steel, dyes and pigments, chrome plating, leather tanning, and wood preservation

Contaminant:
Any physical, chemical, biological, or radiological substance or matter in air, water, or soil that can have adverse health effects.

Cotinine:
A major metabolite of nicotine found in blood and urine. Currently regarded as the best biomarker for exposure of both active smokers and of nonsmokers to environmental tobacco smoke.

Criteria Pollutant:
One of the six pollutants for which EPA is required to set National Ambient Air Quality Standards to protect human health and welfare. Criteria pollutants include ozone (ground-level), carbon monoxide, particulate matter, sulfur dioxide, lead, and nitrogen oxides. They are called "criteria" pollutants because the Clean Air Act required EPA to describe the criteria for setting or revising standards.

Deciliter:
One-tenth of a liter (0.1 liter).

Diesel:
A petroleum-based fuel. Diesel exhaust is an important source of particulates and other pollutants that adversely affect human health.

Glossary of Terms

Dioxins:
A group of harmful chemical compounds that are released into the air from combustion processes such as commercial or municipal waste incineration and from burning fuels such as wood, coal, or oil.

Disinfection Byproducts:
Organic and inorganic compounds that often result from the reaction between a disinfectant and naturally occurring materials in water; chloroform is a commonly found example.

Down Syndrome:
A genetic condition usually caused by having an extra copy of the 21st chromosome. Also called trisomy 21.

Environmental Tobacco Smoke:
Mixture of smoke exhaled by a smoker and the smoke from the burning end of the smoker's cigarette, pipe, or cigar. Also known as secondhand smoke. Environmental tobacco smoke is an important indoor air pollutant.

Epidemiological Studies:
Studies that research the incidence, distribution, and control of disease in a population.

Ewing's Sarcoma:
A type of bone cancer that usually forms in the middle (shaft) of large bones

Exacerbation of Asthma:
Increase in the frequency or severity of asthma attacks or symptoms in individuals who have asthma.

Exposure:
Human contact with environmental contaminants in media including air, water, soil, and food.

Formaldehyde:
A colorless, pungent-smelling gas; an important hazardous air pollutant. High concentrations may trigger attacks in people with asthma. Sources include environmental tobacco smoke and other combustion sources; pressed wood products (such as particle board); and certain textiles, foams, and glues.

Gastrointestinal:
Relating to, affecting, or including the stomach and/or intestine.

Germ Cell Tumor:
A type of tumor found in the ovaries or testicles.

Gonadal Tumor:
Tumor specific to the gonads.

Ground level ozone:
Ground-level ozone (smog) is formed by a chemical reaction between volatile organic pollutants (VOCs) and oxides of nitrogen (NO_x) in the presence of sunlight. Ozone concentrations can reach unhealthy levels when the weather is hot and sunny with little or no wind. Ozone at the ground level causes adverse effects on lung function and other adverse respiratory effects. It is one of the six "criteria" pollutants for which EPA has adopted National Ambient Air Quality Standards.

Hazardous Air Pollutants:
Air pollutants identified in the Clean Air Act Amendments of 1990 as reasonably expected to cause or contribute to irreversible illness or death. Such pollutants include asbestos, beryllium, mercury, benzene, coke oven emissions, radionuclides, and vinyl chloride. A total of 188 hazardous air pollutants are listed in section 112(b) of the Clean Air Act, as amended in 1990. There are no ambient air quality standards for these pollutants.

Heavy Metals:
Metallic elements with high atomic weights, e.g., mercury, chromium, cadmium, arsenic, and lead; can damage living things at low concentrations.

Hodgkin's Lymphoma:
A cancer of the lymphatic system that is characterized by enlargement of lymph nodes, the spleen, or other lymphatic tissue.

Hypospadias:
A birth defect found in boys in which the urinary tract opening is not located properly at the tip of the penis.

Immunodeficiency:
A disorder in which the immune system is reduced or absent.

Ionizing Radiation:
Radiation that can strip electrons from atoms, i.e., alpha, beta, and gamma radiation. High doses can causes massive tissue damage; lower doses can lead to cancer and harmful genetic mutations.

Glossary of Terms

Leukemia:
A cancer in which the body produces a large number of abnormal blood cells.

Lymphocytic Leukemia:
The most common form of childhood leukemia, also known as lymphoblastic leukemia. In this disease, the bone marrow produces large quantities of immature lymphocytes (white blood cells).

Lymphoma:
Lymphomas are tumors in the lymph system, which is responsible for fighting diseases in the body and is part of the immune system.

Maximum Contaminant Level (MCL):
The highest level of a contaminant that is allowed in drinking water as delineated by the National Primary Drinking Water Regulations. These levels are based on consideration of health risks, technical feasibility of treatment, and cost-benefit analysis.

Media:
Specific environments such as air, water, food, and soil.

Mercury:
A heavy metal that is highly toxic if breathed or swallowed. The organic form of mercury, methylmercury, bioaccumulates in ecosystems and can cause adverse effects on children exposed before birth or adults at higher concentrations. The largest human-generated source of mercury emissions in the United States is the burning of coal. Other sources include the combustion of waste and industrial processes that use mercury.

Methemoglobinemia:
A condition that reduces the ability of the blood to transport oxygen throughout the body for essential metabolism; it is due to the replacement of hemoglobin with methemoglobin in the blood. A small amount of methemoglobin is present in the blood normally, but injury or toxic agents—such as nitrites—convert a larger proportion of hemoglobin into methemoglobin.

Methylmercury:
An organic form of mercury, created from metallic or elemental mercury by bacteria in sediments. Methylmercury is easily absorbed into the living tissue of aquatic organisms and is not easily eliminated. Therefore, it accumulates in organisms at the top of food chains such as tuna or humans. It can cause adverse effects in children exposed before or after birth.

Microgram (µg):
One-millionth of a gram.

µg/dL:
Microgram per deciliter.

Microorganisms:
Tiny living organisms that can be seen only with the aid of a microscope. Some microorganisms can cause acute health problems when consumed in drinking water. Also known as microbes.

Monitoring and Reporting Violation:
Violation of monitoring and reporting requirements that specify how and when water must be tested for the presence of contaminants as defined by the Safe Drinking Water Act.

Mortality:
The number of deaths in a population, or death rate.

Myeloid Leukemia:
One form of cancer of the blood-forming tissue, primarily the bone marrow and lymph nodes.

National Ambient Air Quality Standards (NAAQS):
Standards established by EPA for maximum allowable concentrations of six "criteria" pollutants in outdoor air. The six pollutants are carbon monoxide, lead, ground-level ozone, nitrogen dioxide, particulate matter, and sulfur dioxide. The standards are set at a level that protects public health with an adequate margin of safety.

National Priorities List:
List of sites under EPA's Superfund program, which investigates and cleans up hazardous sites nationwide. Sites on the National Priorities List have undergone preliminary assessment and site inspection and have been determined to require remediation due to potential threats to persons living or working near the site.

Neuroblastomas:
Cancer that arises in immature nerve cells and affects mostly infants and children.

Nitrates and Nitrites:
Nitrogen-oxygen chemical units that combine with various organic and inorganic compounds. Once taken into the body, nitrates are converted into nitrites. The greatest use of nitrates is as a fertilizer. Other sources include animal manure and human sewage.

Glossary of Terms

Nitrogen Dioxide (NO_2):
A chemical that results from nitric oxide combining with oxygen in the atmosphere; a major component of photochemical smog. One of the six "criteria" pollutants for which EPA has set national ambient air quality standards.

Nitrogen Oxides:
A family of highly reactive gases (including nitrogen dioxide, above) that form when fuel is burned at high temperatures. Emitted principally from motor vehicle exhaust and stationary sources such as electric power plants and industrial boilers.

Non-Hodgkin's Lymphoma:
A group of cancers of the lymphoid system.

Oral Cleft Defects:
An abnormal opening in a structure around the mouth and face. Clefts may occur in the lip, the roof of the mouth (hard palate), or the tissue in the back of the mouth (soft palate).

Organophosphate Pesticides:
A group of approximately 40 closely related pesticides that affect functioning of the nervous system. Examples include chlorpyrifos, phosmet, and methyl parathion.

Ozone:
A gas that results from complex chemical reactions between nitrogen dioxide and volatile organic compounds; the major component of smog. Ozone at the ground level is one of the six "criteria" pollutants for which EPA has established national ambient air quality standards.

Particulate Matter:
Particles in the air, such as dust, dirt, soot, smoke, and droplets. Small particles (PM-10 or PM-2.5) have significant effects on human health. Particulate matter is one of the six "criteria" pollutants for which EPA has established national ambient air quality standards.

Plasticizers:
Small, often volatile molecules that are added to hard, stiff plastics to make them softer and more flexible.

Polychlorinated Biphenyls (PCBs):
A group of toxic, persistent chemicals used in electrical transformers and capacitors for insulating purposes, and in gas pipeline systems as a lubricant. The sale and new use of PCBs were banned by law in 1979 although large reservoirs of PCBs remain in the environment.

Poverty Level:
An income level below which an individual or family is considered poor. The U.S. Census Bureau defines poverty level based on a set of money income thresholds that vary by family size and composition. If a family's total income is less than that family's threshold, then that family, and every individual in it, is considered poor. The Census Bureau updates its poverty thresholds annually. In 2000, a family of two adults and two children with total income below $17,463 was considered below the poverty level. Tables showing the Census Bureau's poverty thresholds are available at http://www.census.gov/hhes/poverty/threshld.html.

Prenatal:
Occurring, existing, or performed before birth.

Radionuclides:
Radioactive isotopes or unstable forms of elements.

Retinoblastomas:
Tumors of the eye.

Respiratory Effects:
Effects on the process of breathing or on the lungs.

Respiratory Mortality:
Death or the death rate due to respiratory illness.

Reference Dose (RfD):
Oral reference dose. EPA defines a reference dose as an estimate, with uncertainty spanning perhaps an order of magnitude, of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

Solvents:
Substances used to dissolve another substance. Some commonly used solvents, such as TCE, are important environmental contaminants.

Sudden Infant Death Syndrome (SIDS):
The sudden and unexpected death of an apparently healthy infant, without an apparent cause.

Sulfur Dioxide (SO_2):
A pungent, colorless, gaseous pollutant formed primarily by the combustion of fossil fuels. One of the six "criteria" pollutants for which EPA has set national ambient air quality standards.

Glossary of Terms

Superfund:
An EPA program to remediate sites contaminated by release of hazardous substances. Activities include establishing a National Priorities List, investigating sites for inclusion on the list, determining their priority, and conducting and/or supervising cleanup and other remedial actions. Superfund is operated under the legislative authority of the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA). Some remedial actions are funded directly by Superfund, through a tax on chemical feedstocks, but the majority are paid for by parties that are liable for the release of the hazardous substances.

Trichloroethylene (TCE):
A stable, low boiling-point colorless liquid, toxic if inhaled. Used as a solvent or metal decreasing agent, and in other industrial applications.

Volatile Organic Pollutants:
Carbon-containing compounds that easily go from a solid to a gaseous form at normal temperatures. Sources include household products such as paints, paint strippers, and other solvents; wood preservatives; aerosol sprays; cleansers and disinfectants; moth repellents and air fresheners; stored fuels and automotive products; hobby supplies; dry-cleaned clothing.

Wilms' Tumor:
A kidney cancer that occurs in children usually younger than 5 years.

APPENDIX A

Data Tables

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Part 1: Environmental Contaminants

Table E1

Percentage of children living in counties in which air quality standards were exceeded

1990-1995

	1990	1991	1992	1993	1994	1995
Ozone one-hour standard	22.7%	25.2%	16.9%	21.1%	19.3%	27.8%
PM-10	8.0%	6.3%	9.6%	2.6%	2.3%	10.0%
Carbon monoxide	9.4%	8.4%	6.1%	5.0%	6.4%	4.9%
Lead	2.2%	6.0%	1.8%	2.1%	1.7%	1.8%
Sulfur dioxide	0.5%	2.1%	0.1%	0.5%	0.1%	0.1%
Nitrogen dioxide	3.7%	3.7%	0.0%	0.0%	0.0%	0.0%
Any standard*	28.0%	31.8%	20.9%	24.2%	23.5%	30.8%

1996-2001

	1996	1997	1998	1999	2000	2001
Ozone one-hour standard	16.5%	18.6%	20.8%	21.7%	13.3%	15.0%
Ozone eight-hour standard	39.1%	38.9%	48.5%	46.9%	27.9%	39.8%
PM-10	1.5%	2.4%	2.0%	2.1%	2.4%	3.2%
PM-2.5					27.2%	25.4%
Carbon monoxide	5.6%	3.7%	4.3%	3.7%	3.8%	0.2%
Lead	1.6%	1.4%	1.6%	0.2%	0.5%	1.0%
Sulfur dioxide	0.1%	0.1%	0.1%	0.1%	0.1%	0.0%
Nitrogen dioxide	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Any standard*	19.8%	21.9%	23.7%	24.0%	15.5%	18.5%

* Does not include ozone eight-hour or PM-2.5 standards.

SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

Addendum 1 to Table E1

Percentage of children living in counties in which air quality standards were exceeded, by race/ethnicity, 1999

All Races/ Ethnicities	White non-Hispanic	Black non-Hispanic	Hispanic	American Indian/ Alaska Native	Asian or Pacific Islander
15.5%	10.6%	16.2%	31.4%	7.6%	24.5%

SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

Addendum 2 to Table E1

Percentage of children living in counties in which air quality standards were exceeded, by family income, 1999

	All Incomes	< Poverty Level	100-200% of Poverty Level	> 200% of Poverty Level
Carbon monoxide	3.7%	4.5%	4.3%	3.2%
Sulfur dioxide	0.1%	0.1%	0.1%	0.1%
Nitrogen dioxide	0.0%	0.0%	0.0%	0.0%
Ozone	21.7%	20.9%	19.4%	22.7%
Lead	0.2%	0.1%	0.2%	0.2%
PM-10	2.1%	2.1%	2.2%	2.0%

SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

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Table E2

Percentage of children's days with good, moderate, or unhealthy air quality

1990-1995

Pollution Level	1990	1991	1992	1993	1994	1995
Good	43.6%	44.2%	47.7%	46.9%	45.7%	47.2%
Moderate	20.6%	21.0%	18.4%	19.2%	20.5%	19.7%
Unhealthy	3.0%	3.0%	2.7%	2.3%	2.3%	2.2%
No Monitoring Data	32.8%	31.8%	31.2%	31.6%	31.5%	30.8%

1996-1999

Pollution Level	1996	1997	1998	1999
Good	48.9%	48.8%	47.1%	46.6%
Moderate	19.1%	19.0%	20.7%	21.9%
Unhealthy	1.7%	1.3%	1.3%	0.9%
No Monitoring Data	30.3%	30.9%	30.9%	30.7%

SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

Addendum 1 to Table E2

Percentage of children's days with good, moderate, or unhealthy air quality, by race/ethnicity, 1999

Pollution Level	White non-Hispanic	Black non-Hispanic	Hispanic	American Indian/ Alaska Native	Asian or Pacific Islander
Good	44.4%	33.4%	51.0%	60.0%	48.9%
Moderate	18.7%	14.6%	22.6%	27.4%	33.2%
Unhealthy	0.6%	0.4%	0.9%	1.3%	1.9%
No Monitoring Data	36.4%	51.6%	25.6%	11.3%	16.0%

SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

Addendum 2 to Table E2

Percentage of children's days with good, moderate, or unhealthy air quality, by family income, 1999

Pollution Level	All Incomes*	< Poverty Level	100-200% of Poverty Level	> 200% of Poverty Level
Good	46.9%	44.9%	42.5%	49.0%
Moderate	22.0%	22.3%	21.4%	22.1%
Unhealthy	0.9%	0.9%	0.9%	0.8%
No Monitoring Data	30.2%	31.9%	35.2%	28.0%

* Values for All Incomes in this table differ from 1999 values in Table E2 due to rounding.

SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

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Table E3a

Long-term trends in annual average concentrations of criteria pollutants

1990-1993

	1990	1991	1992	1993
PM-10, percent of annual standard	63.7%	63.8%	56.8%	55.8%

1994-1997

	1994	1995	1996	1997
PM-10, percent of annual standard	56.3%	54.0%	52.4%	52.6%
Nitrogen dioxide, percent of annual standard			37.3%	34.9%
Sulfur dioxide, percent of annual standard			6.5%	5.6%

1998-2000

	1998	1999	2000
PM-10, percent of annual standard	51.2%	53.9%	53.3%
Nitrogen dioxide, percent of annual standard	35.3%	33.9%	
Sulfur dioxide, percent of annual standard	5.4%	5.2%	

SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

Table E3b

Number of children living in counties with high annual averages of PM-10

1990-1995

	1990	1991	1992	1993	1994	1995
Exceeding 80% of the long-term standard	5,978,059	6,347,396	4,671,899	4,350,278	1,432,268	3,019,285
Exceeding the long-term standard	1,844,770	3,424,292	169,004	536,520	435,493	131,590

1996-2000

	1996	1997	1998	1999	2000
Exceeding 80% of the long-term standard	2,464,947	2,074,549	165,431	2,029,422	1,830,579
Exceeding the long-term standard	60,243	788,945	0	1,017,791	874,734

SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Aerometric Information Retrieval System

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Table E4

Percentage of children living in counties where estimated hazardous air pollutant concentrations were greater than health benchmarks in 1996

Health Benchmark	
Cancer, one in 100,000	100%
Cancer, one in 10,000	18%
Other health effects	95%

SOURCE: U.S. Environmental Protection Agency, National Air Toxics Assessment

Addendum 1 to Table E4

Percentage of children living in counties where hazardous air pollutant concentrations were greater than health benchmarks in 1996, by family income

Health Benchmark	All Incomes	< Poverty Level	100-200% of Poverty Level	> 200% of Poverty Level
Cancer, one in 100,000	100%	100%	100%	100%
Cancer, one in 10,000	18%	22%	17%	17%
Other health effects	95%	94%	93%	96%

SOURCE: U.S. Environmental Protection Agency, National Air Toxics Assessment

Addendum 2 to Table E4

Percentage of children living in counties where hazardous air pollutant concentrations were greater than health benchmarks in 1996, by race/ethnicity

Health Benchmark	All Races/ Ethnicities	White non-Hispanic	Black non-Hispanic	American Indian/ Alaska Native	Asian or Pacific Islander	Hispanic
Cancer, one in 100,000	100%	100%	100%	100%	100%	100%
Cancer, one in 10,000	18%	12%	28%	8%	34%	31%
Other health effects	95%	94%	99%	87%	99%	95%

SOURCE: U.S. Environmental Protection Agency, National Air Toxics Assessment

Table E5

Percentage of homes with children under 7 where someone smokes regularly

1994	1996	1999
28.7%	27.6%	19.0%

SOURCE: U.S. Environmental Protection Agency, Office of Air and Radiation, Indoor Environments Division, Surveys on Radon Awareness and Environmental Tobacco Issues

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Table E6

Percentage of children living in areas served by public water systems that exceeded a drinking water standard or violated treatment requirements

1993-1997

Type of standard violated	**1993**	**1994**	**1995**	**1996**	**1997**
Lead and copper*	2.2%	0.9%	1.4%	1.6%	1.7%
Microbial contaminants	8.3%	7.5%	4.1%	4.3%	3.6%
Chemical and radiation	4.7%	4.7%	2.2%	1.8%	2.4%
Nitrate/nitrite	0.23%	0.12%	0.25%	0.20%	0.37%
Treatment and filtration	10.7%	8.1%	4.5%	3.7%	3.6%
Any health-based violations	20.2%	15.5%	12.0%	10.7%	10.7%

1998-1999

Type of standard violated	**1998**	**1999**
Lead and copper*	1.6%	1.5%
Microbial contaminants	2.8%	2.5%
Chemical and radiation	1.2%	1.0%
Nitrate/nitrite	0.17%	0.21%
Treatment and filtration	3.4%	3.0%
Any health-based violations	8.6%	8.0%

* Lead and copper represents the lead and copper rule, which is a set of standards and implementation measures.

SOURCE: U.S. Environmental Protection Agency, Office of Water, Safe Drinking Water Information System

Table E7

Percentage of children living in areas with major violations of drinking water monitoring and reporting requirements

1993-1997

Type of standard violated	**1993**	**1994**	**1995**	**1996**	**1997**
Lead and copper	11.3%	6.7%	5.3%	5.2%	5.8%
Microbial contaminants	2.2%	2.6%	2.1%	1.6%	2.0%
Chemical and radiation	8.1%	5.8%	5.5%	4.8%	3.5%
Treatment and filtration	1.6%	0.6%	0.4%	0.5%	0.3%
Any major violation	21.6%	14.2%	11.7%	10.9%	10.9%

1998-1999

Type of standard violated	**1998**	**1999**
Lead and copper	5.5%	5.4%
Microbial contaminants	1.9%	1.4%
Chemical and radiation	3.8%	2.8%
Treatment and filtration	0.5%	1.0%
Any major violation	10.6%	9.9%

SOURCE: U.S. Environmental Protection Agency, Office of Water, Safe Drinking Water Information System

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Table E8

Percentage of fruits, vegetables, and grains with detectable residues of organophosphate pesticides

1994	1995	1996	1997	1998	1999	2000	2001
20.8%	24.4%	29.4%	28.8%	22.1%	24.4%	23.2%	19.1%

SOURCE: U.S. Department of Agriculture, Pesticide Data Program

Table E9

Percentage of children residing within one mile of a Superfund site

	1990	1992	1994	1996	1998	2000
All Superfund sites	1.2%	1.2%	1.3%	1.3%	1.3%	1.3%
All Superfund sites not yet cleaned up or controlled	1.2%	1.1%	1.1%	1.0%	1.0%	0.8%

SOURCE: U.S. Environmental Protection Agency, Superfund NPL Assessment Program (SNAP) Database

Part 2: Body Burdens

Table B1

Concentrations of lead in blood of children ages 5 and under

	Blood lead concentrations (µg/dL)			
	1976-1980	1988-1991	1992-1994	1999-2000
50th percentile	15.0	3.5	2.6	2.2
90th percentile	25.0	9.4	7.1	4.8

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

Table B2

Median concentrations of lead in blood of children ages 1-5, by race/ethnicity and family income, 1999-2000

	Blood lead concentrations (µg/dL)				
	All Incomes	< Poverty Level	100-200% of Poverty Level	> 200% of Poverty Level	Unknown Income
All Races/Ethnicities	2.2	2.8	1.9	1.9	2.9
White non-Hispanic	2.1	2.8	1.7	2.0	3.2
Black non-Hispanic	2.8	3.6	2.6	2.2	2.7
Hispanic	2.0	2.4	1.7	1.6	2.3

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

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Table B3

Distribution of concentrations of lead in blood of children ages 1-5, 1999-2000

Blood lead concentrations (µg/dL)

< 1	1-2	2-3	3-4	4-5	5-6	6-7	> 7
10%	36%	24%	14%	6%	3%	2%	5%

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

Table B4

Distribution of concentrations of mercury in blood of women of child-bearing age, 1999-2000

Blood mercury concentrations (parts per billion)

< 1	1-2	2-3	3-4	4-5	5-6	6-7	
51%	23%	8%	4%	4%	2%	2%	
7-8	**8-9**	**9-10**	**10-11**	**11-12**	**12-13**	**13-14**	**> 14**
1%	1%	0%	1%	1%	1%	0%	1%

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

Table B5

Concentrations of cotinine in blood of children

	Serum cotinine concentrations (ng/mL)	
	1988-1991	1999-2000
90th percentile	2.16	1.78
50th percentile	0.25	0.11

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

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Addendum to Table B5

Median concentrations of cotinine in blood of children ages 4-17, by race/ethnicity and family income, 1988-1991

	Serum cotinine concentrations (ng/mL)				
	All incomes	< Poverty Level	100-200% of Poverty Level	> 200% of Poverty Level	Unknown Income
All races/ethnicities	0.25	0.66	0.34	0.16	0.30
White non-Hispanic	0.23	1.15	0.38	0.16	0.37
Black non-Hispanic	0.59	0.80	0.77	0.24	0.47
Hispanic	0.19	0.21	0.17	0.12	0.19

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

Part 3: Childhood Illnesses

Table D1

Percentage of children with asthma

1980-1985

	1980	1981	1982	1983	1984	1985
Percentage of children with asthma in the past 12 months	3.6%	3.7%	4.1%	4.5%	4.3%	4.8%

1986-1991

	1986	1987	1988	1989	1990	1991
Percentage of children with asthma in the past 12 months	5.1%	5.3%	5.0%	6.1%	5.8%	6.4%

1992-1996

	1992	1993	1994	1995	1996
Percentage of children with asthma in the past 12 months	6.3%	7.2%	6.9%	7.5%	6.2%

1997-2001*

	1997	1998	1999	2000	2001
Children ever diagnosed with asthma and having an asthma attack in the past 12 months	5.4%	5.3%	5.3%	5.5%	5.7%
Children ever diagnosed with asthma	11.4%	12.1%	10.8%	12.3%	12.6%

* Note: The survey questions for asthma changed in 1997; data before 1997 cannot be directly compared to data in 1997 and later.
SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

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Table D2

Percentage of children having an asthma attack in the previous 12 months, by race/ethnicity and family income, 1997-2000

	All Incomes	< Poverty Level	100-200% of Poverty Level	> 200% of Poverty Level	Unknown Income
All races/ethnicities	5.4%	6.4%	5.5%	5.3%	4.9%
White non-Hispanic	5.2%	6.1%	5.5%	5.1%	4.7%
Black non-Hispanic	7.2%	8.5%	7.2%	6.3%	6.5%
Hispanic	4.6%	5.0%	3.9%	5.2%	4.3%

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

Table D3

Children's emergency room visits for asthma and other respiratory causes

1992-1997

	Rate per 10,000 children					
	1992	1993	1994	1995	1996	1997
All asthma and other respiratory causes	369.1	406.6	392.4	357.2	356.4	374.6
Acute upper respiratory infections	221.9	248.7	246.8	227.4	206.4	214.0
Asthma	97.6	107.1	105.1	92.6	114.4	112.1
Acute bronchitis	49.6	50.8	40.5	37.1	35.6	48.5

1998-1999

	1998	1999
All asthma and other respiratory causes	389.2	378.7
Acute upper respiratory infections	218.9	239.0
Asthma	124.4	104.5
Acute bronchitis	45.9	35.1

Note: Respiratory infections are ICD-9 codes 464 and 465, acute bronchitis is ICD-9 code 466, and asthma is ICD-9 code 493.
SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Hospital Ambulatory Medical Care Survey

Addendum to Table D3

Children's emergency room visits for asthma and other respiratory causes, by race/ethnicity, 1997-1999

	Rate per 10,000 children			
	1997	1998	1999	1997-1999
White non-Hispanic	298.4	311.9	317.7	309.3
Black non-Hispanic	826.5	811.7	663.9	767.3
Hispanic	*	*	*	384.6

* Annual estimates for Hispanic ethnicity are unreliable for asthma.
SOURCE: Centers for Disease Control and Prevention, National Hospital Ambulatory Medical Care Survey

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Table D4

Children's hospital admissions for asthma and other respiratory causes

1980-1985

	Rate per 10,000 children					
	1980	**1981**	**1982**	**1983**	**1984**	**1985**
All asthma and other respiratory causes	54.7	56.1	55.3	61.4	50.4	56.6
Acute upper respiratory infections	17.6	17.7	12.7	19.4	11.4	14.1
Acute bronchitis	16.2	16.3	16.9	18.3	13.5	17.1
Asthma	21.0	22.2	25.7	23.6	25.5	25.4

1986-1991

	1986	**1987**	**1988**	**1989**	**1990**	**1991**
All asthma and other respiratory causes	49.9	52.3	52.5	58.8	54.5	62.8
Acute upper respiratory infections	10.0	12.8	9.5	11.8	8.3	11.4
Acute bronchitis	12.6	13.5	14.6	19.1	17.8	20.7
Asthma	27.3	25.9	28.5	27.9	28.4	30.7

1992-1997

	1992	**1993**	**1994**	**1995**	**1996**	**1997**
All asthma and other respiratory causes	61.1	56.3	54.2	66.1	57.5	68.0
Acute upper respiratory infections	7.2	9.0	7.1	8.2	5.9	7.7
Acute bronchitis	22.9	21.7	20.0	24.7	21.3	27.3
Asthma	31.0	25.6	27.1	33.1	30.3	33.0

1998-1999

	1998	**1999**
All asthma and other respiratory causes	50.3	66.4
Acute upper respiratory infections	5.9	8.4
Acute bronchitis	19.5	29.2
Asthma	24.9	28.8

Note: Respiratory infections are ICD-9 codes 464 and 465, acute bronchitis is ICD-9 code 466, and asthma is ICD-9 code 493.
SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Hospital Discharge Survey

Addendum to Table D4

Children's hospital admissions for asthma and other respiratory causes, by race/ethnicity, 1997-1999*

	Rate per 10,000 children			
	1997	**1998**	**1999**	**1997-1999**
White	45.6	34.3	45.5	41.8
Black	107.7	78.6	99.2	95.2

* Estimates for ethnicity not available. Race categories include children of Hispanic ethnicity.
SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Hospital Discharge Survey

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Table D5

Cancer incidence and mortality for children under 20

1975-1980

	Rate per million children					
	1975	**1976**	**1977**	**1978**	**1979**	**1980**
Incidence	128	141	141	144	145	142
Mortality	51	51	50	45	46	46

1981-1986

	1981	**1982**	**1983**	**1984**	**1985**	**1986**
Incidence	138	144	144	154	157	157
Mortality	44	45	43	39	38	38

1987-1992

	1987	**1988**	**1989**	**1990**	**1991**	**1992**
Incidence	152	151	166	155	162	160
Mortality	36	35	35	34	34	33

1993-1998

	1993	**1994**	**1995**	**1996**	**1997**	**1998**
Incidence	161	156	158	162	156	161
Mortality	33	32	30	29	29	28

SOURCE: Incidence data from National Cancer Institute, Surveillance, Epidemiology, and End Results Program; mortality data from Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System

Addendum 1 to Table D5

Cancer incidence for children under 20 by race/ethnicity and gender, 1994-1998

	Rate per million children	
	Male	**Female**
All races/ethnicities	167	150
White non-Hispanic	172	156
Black non-Hispanic	133	117
Hispanic	150	141
American Indian/Alaska Native	82	62
Asian or Pacific Islander	150	132

SOURCE: National Cancer Institute, Division of Cancer Control and Population Sciences. Surveillance, Epidemiology, and End Results Program 1994-1998

Addendum 2 to Table D5

Age-adjusted cancer mortality rates for children under 20 by race/ethnicity and gender, 1994-1998

	Rate per million children	
	Male	**Female**
All races/ethnicities	33	26
White non-Hispanic	34	26
Black non-Hispanic	33	29
Hispanic	35	27
American Indian/Alaska Native	25	19
Asian or Pacific Islander	30	24

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System

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Table D6

Cancer incidence for children under 20 by type

	Rate per million children				
	1974-78	1979-83	1984-88	1989-93	1994-98
Acute lymphoblastic leukemia	23.7	24.9	27.6	28.2	28.3
Acute myeloid leukemia	5.2	4.9	3.8	5.1	4.8
Central nervous system tumors	23.2	22.2	27.9	30.1	27.3
Hodgkin's lymphoma	13.9	14.2	14.1	14.1	12.8
Non-Hodgkin's lymphoma	9.1	9.5	10.4	10.2	11.2
Thyroid carcinoma	4.8	4.8	5.1	5.1	5.4
Malignant melanoma	3.9	4.3	5.4	6.5	6.2
Germ cell tumors	8.3	9.9	9.7	11.7	11.7
Soft tissue sarcomas	10.0	10.7	10.9	11.4	11.6
Osteosarcoma	3.8	4.8	5.0	5.0	5.5
Ewing's sarcoma	2.4	3.5	3.4	3.0	3.1
Neuroblastoma	7.3	7.2	7.9	7.7	8.0
Wilms' tumor	5.4	6.5	5.6	6.3	6.5
Hepatoblastoma	0.7	0.7	1.1	1.2	1.4

SOURCE: National Cancer Institute, Division of Cancer Control and Population Sciences. Surveillance, Epidemiology, and End Results Program 1994-1998

Addendum to Table D6

Cancer incidence for children under 20 by age and type, 1994-1998

	Rate per million children			
	Ages 0-4	Ages 5-9	Ages 10-14	Ages 15-19
Lymphocytic leukemia	61.0	30.6	18.4	14.9
Acute non-lymphocytic leukemia	9.7	4.3	6.5	7.8
Hodgkin's lymphoma	0.9	3.7	11.8	32.0
Non-Hodgkin's lymphoma	3.6	5.9	7.7	13.2
CNS and miscellaneous intracranial and intraspinal neoplasms	34.5	29.7	25.0	19.2
Neuroblastoma and ganglioneuroblastoma	25.7	3.2	0.8	0.4
Wilms' tumor	18.8	4.8	0.8	0.3
Hepatic tumors	4.6	0.6	0.5	1.2
Osteosarcoma	0.2	2.6	7.9	8.9
Ewing's sarcoma	0.6	2.2	4.2	4.7
Soft tissue sarcomas	11.1	7.9	10.5	15.0
Germ cell, trophoblastic, other gonadal neoplasms	7.2	2.1	7.2	28.1
Epithelial and unspecified	3.1	3.3	11.6	40.5

SOURCE: National Cancer Institute, Division of Cancer Control and Population Sciences. Surveillance, Epidemiology, and End Results Program 1994-1998

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Table D7

Children reported to have mental retardation, by race/ethnicity and family income, 1997-2000

	Cases per 1,000 children
All	6
White non-Hispanic	6
Black non-Hispanic	10
Hispanic	5
< Poverty Level	12
100-200% of Poverty Level	10
> 200% of Poverty Level	4
Unknown Income	6

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

Part 4: Emerging Issues

Table EI1

Number of states with advisories for methylmercury in non-commercial fish

	1995	2000
Freshwater advisories	32	37
Coastal and estuarine advisories	7	13
Statewide advisories	5	12

SOURCE: U.S. Environmental Protection Agency, Office of Water. National Listing of Fish and Wildlife Consumption Advisories

Table EI2

Percentage of children ages 5-17 reported to have attention-deficit/hyperactivity disorder, by race/ethnicity and family income, 1997-2000

	All Incomes	< Poverty Level	100-200% of Poverty Level	> 200% of Poverty Level	Unknown Income
All races/ethnicities	6.7%	7.7%	7.5%	6.9%	4.8%
White non-Hispanic	8.0%	13.6%	9.3%	7.5%	6.1%
Black non-Hispanic	5.1%	5.6%	6.5%	4.9%	3.4%
Hispanic	3.8%	3.5%	3.9%	5.1%	2.3%
American Indian/Alaska Native	6.9%				

SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health Interview Survey

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Appendix A: Data Tables

Part 5: Special Features

Table S1

Percentage of California public elementary schools with lead paint and some deterioration of paint, 1994-1997

	Schools Built Before 1940	**Schools Built 1940-1959**	**Schools Built 1960-1979**	**Schools Built 1980-1995**	**All Schools**
Any detectable lead	72.2%	46.3%	20.5%	3.1%	37.4%
≥ 600 ppm	72.2%	46.3%	18.2%	3.1%	36.9%
≥ 5,000 ppm	63.9%	41.8%	13.6%	0%	31.8%

SOURCE: California Department of Health Services, Childhood Lead Poisoning Prevention Branch. Lead Hazards in California's Public Elementary Schools and Child Care Facilities, April 1998

Table S2

Percentage of California public elementary schools with lead in soils, 1994-1997

	Schools Built Before 1940	**Schools Built 1940-1979**	**Schools Built 1980-1995**	**All Schools**
Schools with detectable lead in soil	100.0%	94.4%	60.5%	89.0%
Schools with soil lead > 400 ppm	29.7%	2.4%	0.0%	7.0%

SOURCE: Childhood Lead Poisoning Prevention Branch, California Department of Health Services. Lead Hazards in California's Public Elementary Schools and Child Care Facilities, April 1998

Table S3

Percentage of California public elementary schools with lead in drinking water, 1994-1997

	Schools Built Before 1940	**Schools Built 1940-1979**	**Schools Built 1980-1995**	**All Schools**
Schools with detectable lead in drinking water	68.6%	52.4%	42.1%	53.3%
Schools with > 15 ppb lead in drinking water	31.4%	16.1%	13.2%	18.3%
Schools with > 15 ppb lead in drinking water at first draw*				15.5%
Schools with > 15 ppb lead in drinking water at second draw*				6.5%

* Data for first- and second-draw samples are available for all schools only.

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Table S4

Frequency of application of pesticides in Minnesota K-12 schools, 1999

	Percentage of schools		
	Cafeteria and kitchen food storage areas	Locker Rooms and gymnasium	Classrooms
Sprayed as needed	34.0%	33.5%	46.7%
Sprayed once a month	16.4%	3.7%	2.7%
Sprayed 1 - 4 times per year	24.0%	15.3%	12.5%
Not sprayed	16.9%	28.8%	25.6%
No answer / other / don't know	8.7%	18.7%	12.5%

SOURCE: C.J. Olson Market Research Inc. for Minnesota Department of Agriculture. *Quantitative Research Regarding Pest Management in Minnesota K-12 Schools.* 1999

Table S5

Number of birth defects in California per 1,000 live births and fetal deaths

	1991-1993	1994-1996	1997-1999
Heart defects	1.80	1.98	1.79
Oral cleft defects	1.60	1.67	1.50
Down Syndrome	1.34	1.28	1.28
Intestinal defects	0.73	0.73	0.56
Limb defects	0.46	0.46	0.38

SOURCE: California Birth Defects Monitoring Program

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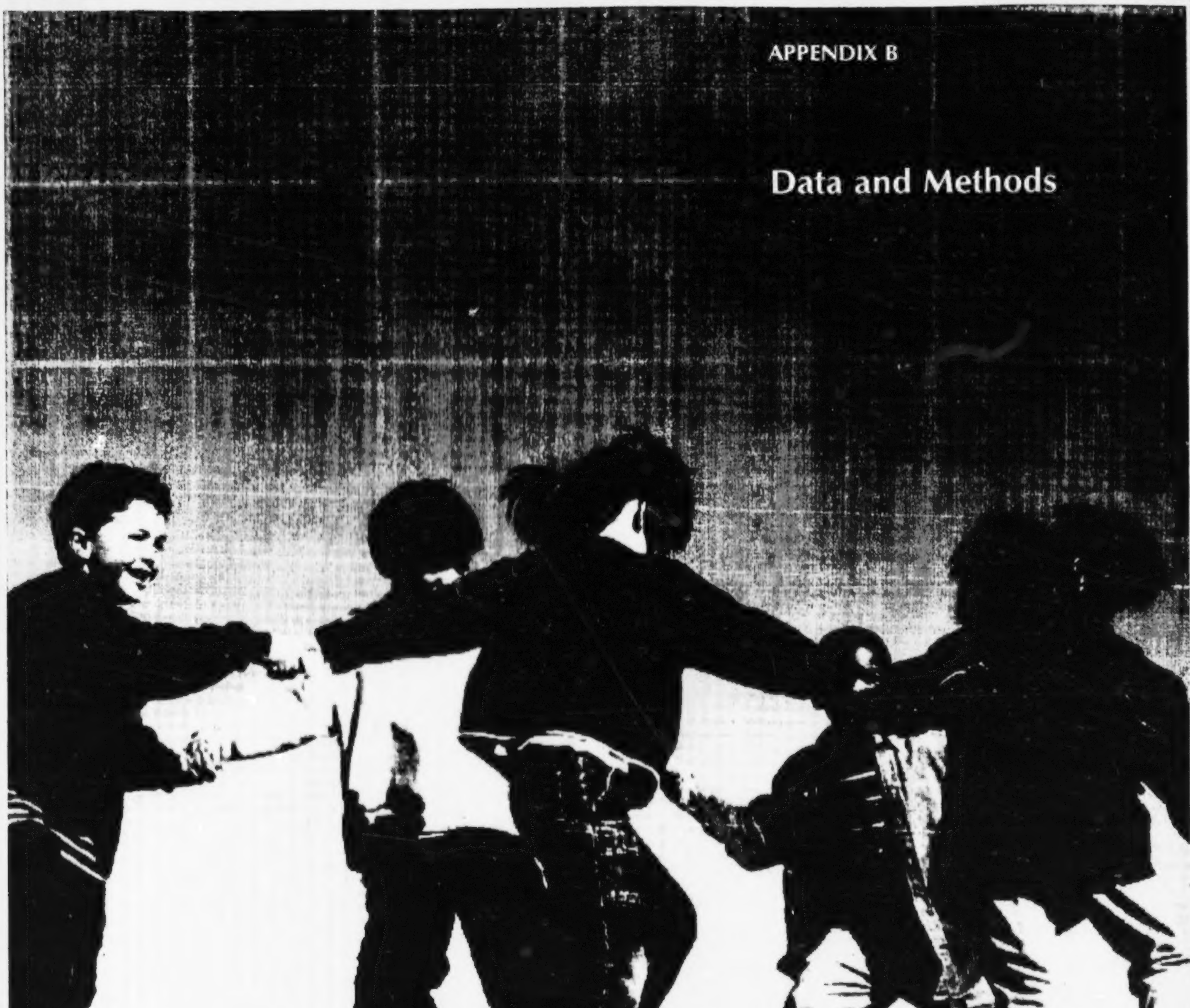
APPENDIX B

Data and Methods



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Common Air Pollutants (Measures E1-E3)

Air Quality Exceedances (Measure E1)

EPA's Office of Air Quality Planning and Standards has set health-based National Ambient Air Quality Standards (NAAQS) for six common pollutants, often referred to as criteria pollutants. These standards are shown in Table 1 below.

Air Quality Exceedances

State and local environmental agencies conduct air monitoring programs to measure concentrations of these pollutants. The individual measurements are submitted to EPA for inclusion in a national database called the Aerometric Information Retrieval System. EPA, as part of its data management system, identifies instances in which levels of air pollutants measured in the air are greater than the air quality standards. Each of these events is called an "exceedance." An exceedance occurs when a measured concentration exceeds a target value that is actually higher than the air quality standard. Concentrations measured in the air must be averaged over a time period set in accordance with the standard for that pollutant. The target values used to identify exceedances are shown in Table 1 below.

Agency Contact:

David Mintz (mintz.david@epa.gov)
U.S. EPA, Office of Air Quality Planning and Standards (OAQPS)
(919) 541-5224

Table 1: National Ambient Air Quality Standards (NAAQS) and the Values Used to Define Exceedances by EPA

Pollutant	Duration of Standard	Standard	Target value to define exceedance
Carbon monoxide	Eight-hour average	9 ppm	9.5 ppm
	One-hour average	35 ppm	Not applicable
Nitrogen dioxide	One year average	0.053 ppm	0.0535 ppm
Ozone	One-hour average[a]	0.12 ppm	0.125 ppm
	Eight-hour average	0.08	0.085 ppm
Lead	Three-month average	1.5 $\mu g/m^3$	1.55 $\mu g/m^3$
Particulate matter under 10 microns	One-day (24 hour) average	150 $\mu g/m$	155 $\mu g/m^3$
	One year average	50 $\mu g/m^3$	Not applicable
Particulate matter under 2.5 microns	One-day (24 hour) average	65 $\mu g/m^3$	Not applicable
	One year average	15 $\mu g/m^3$	Not applicable
Sulfur dioxide	One-day (24 hour) average	0.14 ppm	0.145 ppm
	One year average	0.03 ppm	Not applicable

[a] The ozone 1-hour standard applies only to areas that were designated non-attainment when the ozone 8-hour standard was adopted in July 1997.

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Methods for Air Quality Exceedances (Measure E1)

Measure E1 uses exceedances reported by EPA based on its Aerometric Information Retrieval System. EPA reported counties that exceeded the various standards on at least one day per year. The measure reports the percentage of children living in these counties, obtained by dividing the number of children in counties exceeding the standards on at least one day per year by the total number of children in the United States. Counties were identified that exceeded the following standards at any time during the year:

- Carbon monoxide: one-hour standard
- Ozone: one-hour standard and eight-hour standard
- PM-10: one-day standard
- PM-2.5: annual standard
- Sulfur dioxide: annual standard
- Nitrogen dioxide: annual standard
- Lead: three month standard

Air Quality Index (Measure E2)

Measure E2 is based on the Air Quality Index (AQI) developed by EPA to report daily air quality. The AQI converts measured pollutant concentrations to a number on a scale from 0 to 500. In general, scores of 100 indicate that a daily standard has been reached.

Agency Contact:
AIRS Hotline
U.S. EPA, Office of Air Quality Planning and Standards (OAQPS)
(800) 334-2405

Long-Term Exposure to Criteria Pollutants (Measure E3)

Measure E3a uses concentrations of air pollutants measured at monitoring stations across the United States and reported in EPA's AIRS system. Measured concentrations at each monitoring station were averaged by month and then by county. Annual values, weighted by population, then were calculated. These annual averages of the measured concentrations were compared with the applicable air quality standards to generate the measure shown on the graph.

The measure shown for PM-10 is the average annual concentration experienced by children in the United States, expressed as a percentage of the annual standard of 50 micrograms per cubic meter (μg/m^3). The measure shown for nitrogen dioxide is the average annual concentration experienced by children, expressed as a percentage of the annual standard of 0.053 parts per million. The measure for sulfur dioxide is the average annual concentration experienced by children, expressed as a percentage of the annual standard of 0.03 parts per million.

Measure E3b uses the same data source for measured concentrations. This measure reports the number of children living in counties with concentrations of PM-10 that exceed the specific levels. The data for measured concentrations, averaged as in the previous measure, were used to identify counties within the stated criteria. Census data were used to calculate the number of children in these counties.

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Hazardous Air Pollutants (Measure E4)

The measure on hazardous air pollutants was developed using information from EPA's National Air Toxics Assessment (NATA) for 1996. As part of NATA, EPA estimated ambient concentrations of hazardous air pollutants for every county in the continental United States. EPA used a computer dispersion model, the Assessment System for Population Exposure Nationwide (ASPEN), to estimate these concentrations. As a key input to the model, EPA compiled a 1996 national emissions inventory of air toxics emissions from outdoor sources. The types of emissions sources in the inventory include major stationary sources (e.g., large waste incinerators and factories), area and other sources (e.g., dry cleaners, small manufacturers, wildfires), and both on-road and non-road mobile sources (e.g., cars, trucks, boats).

Although computer modeling of hazardous air pollutant concentrations necessarily requires simplifying assumptions and introduces significant uncertainties, no other method is available for assessing air toxics concentrations nationally. Direct measurements of ambient air toxics concentrations are available for only a subset of air toxics in relatively few locations. In order to understand the limitations of the modeled ambient concentration estimates, EPA compared these estimates to available monitoring data from 1996 for seven pollutants as a "reality check." The comparisons generally show that the model estimates are lower than the monitored concentrations for these pollutants.

EPA used 1996 data because emissions inventories from that year are the most complete and up-to-date available. The 33 air pollutants analyzed in NATA were identified as priority pollutants in EPA's Integrated Urban Air Toxics Strategy. This set includes 32 air toxics that are a subset of EPA's list of 188 toxic air pollutants plus diesel particulate matter, which is used as a surrogate measure of diesel exhaust. EPA has determined that diesel exhaust is likely to be carcinogenic to humans by inhalation at environmental levels of exposure and has listed it as a mobile source air toxic.

More information on NATA is available at http://www.epa.gov/ttn/atw/nata/

Agency Contact:

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(919) 541-5362

Methods for Hazardous Air Pollutants (Measure E4)

Data

Ambient concentrations: Average concentrations of 33 hazardous air pollutants (HAPs) in ambient (outdoor) air for each county in the continental United States were obtained from EPA's National Air Toxics Assessment (NATA). These values are computer-generated estimates of the annual ambient concentrations of the HAPs for 1996. The modeled concentrations, along with more information, are available at http://www.epa.gov/ttn/atw/nata/

Dose-response information: EPA's risk assessments for potentially carcinogenic HAPs typically provide a unit risk estimate (URE), which is an estimate of the excess cancer risk resulting from a lifetime of continuous exposure to a pollutant at a concentration of one microgram per cubic meter (1 $\mu g/m^3$) in air. UREs are estimated by extrapolation of data from laboratory animal studies, or in some cases, human studies (typically of workers who are exposed on the job). Many of the UREs are considered "upper bound," meaning they are an upper estimate of risks from a given exposure level. For several of the more important hazardous air pollutants, including chromium

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and benzene, the cancer risk estimates are based on the statistical best fit to human data, and therefore are less conservative than estimates based on statistical upper confidence limits developed from animal data.

The assessments also provide a reference concentration (RfC) for effects other than cancer, which is an estimate of the concentration in air that is likely to be without appreciable risks of deleterious effects during a lifetime. UREs and RfCs for pollutants assessed in NATA were compiled and discussed in Health Effects Information Used in Cancer and Noncancer Risk Characterization for the NATA 1996 National-Scale Assessment, available at: http://www.epa.gov/ttn/atw/nata/natsa4.html (click on "Health Effects Criteria"). The values from that document were used for this analysis.

Population data: U.S. Census estimates of the number of children, ages 0-17, in each county in the continental United States were obtained for 1996.

Analysis: Cancer Risk Benchmarks
The lifetime cancer risks posed by HAPs in each county were calculated by multiplying the ambient concentration of each HAP by the inhalation unit risk estimate (URE) of that HAP. The risk estimates for all modeled HAPs with cancer unit risk estimates then were summed together to provide a combined cancer risk estimate. The counties for which this value exceeded 1-in-100,000 and 1-in-10,000 were identified, producing two lists of counties. For each list of counties, the number of children ages 0-17 in the identified counties was summed together. The resulting value then was divided by the number of children ages 0-17 in all counties in the continental United States, yielding the percentage of children living in counties where the concentrations of carcinogenic hazardous air pollutants exceeded the two benchmark cancer risk levels.

Analysis: Benchmark for Other Health Effects
A hazard quotient (HQ) was calculated for each HAP with a reference concentration (RfC). The HQ is equal to the modeled ambient concentration divided by the RfC. An HQ greater than one indicates that the concentration of a HAP is greater than that HAP's reference concentration. Counties in which the HQ for any HAP exceeded one were identified. The number of children ages 0-17 in the identified counties was summed together. The resulting value was then divided by the number of children ages 0-17 in all counties in the continental United States to yield the percentage of children living in counties where the concentration of one or more hazardous air pollutants exceeded the health benchmark for effects other than cancer.

Indoor Air Pollutants (Measure E5)

Surveys on Radon Awareness and Environmental Tobacco Smoke Issues

In 1994 and 1996, EPA's Indoor Environments Division commissioned a commercial contractor, Survey Communications, Inc., to conduct surveys on radon awareness and environmental tobacco smoke issues. Approximately 31,000 households in the 50 states were contacted in 1994 and 1996. All interviews were conducted by telephone using a random digit dialing sampling methodology. Both the 1994 and the 1996 surveys asked whether the household included any children under the age of 7. In addition, they asked the following:

- Does anyone in your household smoke cigarettes, cigars, or a pipe?
- Do you allow anyone to smoke in your home on a regular basis?

In the 1994 survey, 6,411 households had children under the age of 7. In the 1996 survey, 6,851 households had children under the age of 7. The percentages of homes with children under the age of 7 in which someone smokes, or in which someone smokes regularly, were obtained by crossing the question on children with the appropriate question on smoking in the household.

In 1999, EPA commissioned the Center for Survey Research and Analysis at the University of Connecticut to conduct a similar but much smaller survey. The results of this survey were based on 1,005 telephone interviews with respondents located in the contiguous 48 states, using a random digit dialing sampling methodology. The survey questions regarding smoking in the home were similar to the questions in the 1994 and 1996 surveys. In the 1999 survey there were 225 households with children under the age of 7. Although the 1999 survey was substantially smaller than the 1994 and 1996 surveys, all three surveys were designed to produce nationally representative samples.

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Drinking Water Contaminants (Measures E6-E7)

Safe Drinking Water Information System (SDWIS)

The Safe Drinking Water Information System (SDWIS) is the national regulatory compliance database for EPA's drinking water program. SDWIS includes information on the nation's 170,000 public water systems and data submitted by states and EPA regions in conformance with reporting requirements established by statute, regulation, and guidance.

EPA sets national standards for drinking water. These requirements take three forms: maximum contaminant levels (MCLs, the maximum allowable level of a specific contaminant in drinking water), treatment techniques (specific methods that facilities must follow to remove certain contaminants), and monitoring and reporting requirements (schedules that utilities must follow to report testing results). States report any violations of these three types of standards to EPA.

Water systems must monitor for contaminant levels on fixed schedules and report to EPA when a maximum contaminant level has been exceeded. States also must report when systems fail to meet specified treatment techniques. More information about the maximum contaminant levels can be found at http://www.epa.gov/OGWDW/mcl.html.

EPA sets minimum monitoring schedules that drinking water systems must follow. These minimum reporting schedules (systems may monitor more frequently) vary by the size of the water system as well as by contaminant. Some contaminants are monitored daily, others need to be checked far less frequently (the longest monitoring cycle is every nine years). For example, at a minimum, drinking water systems will monitor continuously for turbidity, monthly for bacteria, and once every four years for radionuclides.

A monitoring and reporting violation occurs when the system did not perform the required testing, take adequate samples, or report a violation as required. Only major monitoring and reporting violations are used in this report.

SDWIS includes data on the total population served by each public water system and the state in which the public water system is located. However, SDWIS does not include the number of children served. The numbers of children served by the public water systems were estimated by determining the ratio of children in the state in which the public water system is located and multiplying the ratio by the number of people served by that public water system.

Appendix B: Data and Methods

For more information see the EPA's SDWIS Web site at http://www.epa.gov/safewater/sdwisfed/sdwis.htm.

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Methods for Drinking Water Contaminants (Measure E6 and E7)

Safe Drinking Water Information System (SDWIS) Data

The SDWIS database was used to examine national compliance with the Safe Drinking Water Act. Three data files were prepared using data from SDWIS:

- Public Water Systems (PWSs): Describes the public water systems in the United States. Includes data fields with unique PWS Identification numbers and an estimate of the total population served by each PWS.
- SDWIS Maximum Contaminant Level (MCL) Violations: Describes PWS MCL violations. Includes specific violation codes and contaminant group data.
- SDWIS Monitoring and Reporting (MR) Violations: Describes PWS monitoring and reporting violations.

Population Data

Census data on county-level population by age for every county in the United States was obtained from the U.S. Census Bureau. The census population under the age of 18 was summed by county for each state. Subsequently, the proportion of individuals under the age of 18 to the total population in each state was calculated. A census data file was generated containing the proportion of children under the age of 18 living in a given state for each year between 1990 and 2000.

Analysis for Measure E6: Percentage of children living in areas served by public water systems that exceeded a drinking water standard or violated treatment requirements

Data files for Public Water Systems (PWSs), MCL violations and the proportion of children residing within each state were linked by PWS identification number, year, and state. The contaminant and violations codes in the SDWIS MCL Violations file described the type of drinking water standard that was violated. Querying these codes (listed below) generated the data in Measure E6.

- All health-based: All SDWIS MCL violations
- Lead and copper: Violation codes 57-63
- Microbial Contaminants: Violation codes 21 and 22
- Chemical and radiation: All applicable contaminant codes*
- Treatment and filtration: Violation codes 41 and 42
- Nitrate and nitrite: Contaminant codes 1038, 1040, and 1041

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* The applicable contaminant and violations codes for chemical and radiation are as follows:

Total trihalomethanes: 2950

continued on following page

Volatile organic chemicals: 2326, 2378, 2380, 2955, 2964, 2968, 2969, 2976, 2977, 2979, 2980, 2981, 2982, 2983, 2984, 2985, 2987, 2989, 2990, 2991, 2992, 2996

Synthetic organic contaminants: 2005, 2010, 2015, 2020, 2031, 2032, 2033, 2034, 2035, 2036, 2037, 2039, 2040, 2041, 2042, 2046, 2050, 2051, 2063, 2065, 2067, 2105, 2110, 2274, 2306, 2383, 2931, 2946, 2959

Nitrate/nitrite: 1038, 1040, 1041

Inorganic chemicals: 1005, 1010, 1015, 1020, 1024, 1025, 1035, 1045, 1074, 1075, 1085, 1094

Radiological contaminants: 4000, 4010, 4101

Analysis for Measure E7: Percentage of children living in areas with major violations of drinking water monitoring and reporting requirements

Data files for public water systems, monitoring and reporting violations, and the proportion of children residing within each state were linked by PWS identification number, year, and state. Querying the violation codes listed below generated the various trend lines of Measure E7:

- Any major violation: Only major violations selected.
- Lead and copper: Violation codes 51-56
- Microbial contaminants: Violation codes 23 and 25
- Chemical and radiation : Violation code 03
- Treatment and filtration: Violation codes 31 and 36

The initial query resulted in a single table containing merged data on MR violation, PWSs, and census information. Because a PWS can have multiple violations of the same MR rule in the same year, duplicates had to be removed in a second query. The second query selected PWSs only once per year if they reported multiple MR violations in the same contaminant category for that year. The total number of people served by a PWS violating the selected MR violation in a given state was then summed and multiplied by the appropriate proportion of children under the age of 18 living in that state during a given year between 1990 and 2000. The estimate of children age 18 and under served by a PWS violating a MR rule was then summed for all 50 states. The percentage of all children was calculated and presented in Measure E7. The estimate assumes an even geographic distribution by county of individuals under the age of 18 in each state.

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Appendix B: Data and Methods

Pesticide Residues (Measure E8)

Pesticide Data Program

The U.S. Department of Agriculture (USDA) has been conducting the Pesticide Data Program (PDP) since 1991 and has published its findings for calendar years 1991 through 2000. PDP continues to focus on the National Academy of Sciences' 1993 recommendation that pesticide residue monitoring programs target foods that are highly consumed by children, and that the analytical testing methods used in these monitoring efforts should be standardized, validated, and subject to strict quality control and quality assurance programs. Since 1994 PDP has modified its commodity testing profile to include not only fresh fruits and vegetables, but also canned and frozen fruits and vegetables, fruit juices, whole milk, wheat, soybeans, oats, corn syrup, peanut butter, and poultry. In 2001, PDP collected and analyzed more than 12,000 food samples. More information is available at http://www.ams.usda.gov/science/pdp.

Each sample of food tested in the PDP is analyzed to determine whether the residues of a variety of different pesticides are present. The number of organophosphate pesticides and metabolites analyzed by PDP has increased from 34 in 1994 to 77 in 2001, and measurement techniques have become more sensitive during that time. In order to maintain comparability across the years 1994 to 2001, the organophosphate detection rates reported in this measure include only detections of the original 34 pesticides included in the PDP at or above the original limits of detection available in 1994.

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Land Contaminants (Measure E9)

Superfund NPL Assessment Program Database

The Superfund NPL Assessment Program (SNAP) is a relational database system containing data for proposed, final, and deleted National Priorities List (NPL) sites. The majority of the information contained in SNAP is the data that support the NPL listing of sites; e.g., Hazard Ranking System (HRS) scoring factors, site narratives, site characteristics, contaminants, locational information, proposed and final Federal Register dates and citations, etc. For the most part, the data contained in SNAP are a snapshot at the time of NPL proposal and listing, although SNAP also contains a minimal amount of data (date and status) on Construction Completions, partial deletions, and deletions. This information allows SNAP to give an accurate overall picture of the status of the NPL on a real-time basis. All of the data contained in SNAP are publicly available information.

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Methods for Land Contaminants (Measure E9)

Superfund Data
Data describing the physical location of sites listed on the Superfund National Priorities List (NPL) were acquired from EPA's Superfund NPL Assessment Program (SNAP) Database (http://www.epa.gov/superfund/sites/query/advquery.htm). Data fields for each site included parameters such as address, latitude, longitude, date of proposed addition to the NPL, and date deleted from the NPL (where applicable). The latitude-longitude values associated with each NPL site represented an estimate of the geographic center of the site. For a given year, all sites on the NPL or proposed for addition to the NPL on or before September 30th were selected. Sites deleted from the NPL prior to or on September 30th of the same year were then removed. Sites were selected for the period between 1990 and 2000 and the results were exported for use in ArcView GIS version 3.2.

Census Data
U.S. Census data from 1990 and 2000 at the census block level were compiled and processed to obtain fields of information not readily available from the U.S. Census Bureau. The variables in this data set include total population, population under 18, and the latitude and longitude of each census block centroid. The census block centroid latitude-longitude data corresponded to a point at the geographic center of each U.S. census block.

GIS Analysis
Biannual Superfund data as well as the 1990 and 2000 U.S. Census block-level demographic data were imported into ArcView GIS version 3.2. The latitude-longitude points for each Superfund site and census block centroid were plotted using the GIS software. For years prior to 2000, 1990 census block centroids falling within a one mile radius of a Superfund site were selected, on a year-by-year basis between 1990 and 1998. The total number of children associated with the selected census block centroids was then calculated and presented as a percentage of the total number of people under the age of 18 living in the United States. For 2000, U.S. Census block-level population data were used when selecting centroids within a one-mile radius of sites on the NPL in 2000. The total number of children living within one mile of a site on the NPL was summed and presented as a percentage of the total number of children under the age of 18 living in the United States.

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Appendix B: Data and Methods

Concentrations of Lead in Blood (Measures B1-B3)

National Health and Nutrition Examination Survey

Data on children's blood lead levels were obtained from the National Health and Nutrition Examination Surveys (NHANES) II and III, and NHANES 1999-2000, conducted by the National Center for Health Statistics. The survey is designed to assess the health and nutritional status of the non-institutionalized civilian population with direct physical examinations and interviews, using a complex multi-stage, stratified, clustered sampling design. Interviewers obtain information on personal and demographic characteristics, including age, household income, and race and ethnicity by self-reporting or as reported by an informant. The first survey, NHANES I, was conducted during the periods 1971-1974 and 1974-1975; NHANES II covered the period 1976-1980; and NHANES III covered the period 1988-1994. Only NHANES II and III, however, contain data on blood lead levels. NHANES II provided blood lead data for children ages 6 months to 5 years; NHANES III provided data on children ages 1-5 years. Descriptions of the survey design, the methods used in estimation, and the general qualifications of the data are presented in the following:

- Plan and Operation of the Second National Health and Nutrition Examination Survey, 1976-80: Programs and Collection Procedures, Series 1, No. 15. Vital and Health Statistics, Hyattsville, MD: National Center for Health Statistics.
- Plan and Operation of the Third National Health and Nutrition Examination Survey, 1988-94: Series 1: Programs and Collection Procedures, No. 32. Vital and Health Statistics, Hyattsville, MD: National Center for Health Statistics.

Starting in 1999, NHANES changed to a continuous survey visiting 15 U.S. locations per year and surveying and reporting for approximately 5,000 people annually. Body burden data from NHANES 1999-2000 are presented in:

- Second National Report on Human Exposure to Environmental Chemicals. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Environmental Health, January 2003.

The percentage of children with blood lead levels greater than 10 µg/dL is influenced by the proportion of nonresponses within each category. Families with incomes below the poverty level had a lower response rate than families with incomes at or above the poverty level. The percentages are thus the best estimates available, but may be biased by the variation of nonresponses by family income.

NHANES Web site: www.cdc.gov/nchs/nhanes.htm

Second National Report on Human Exposure to Environmental Chemicals: www.cdc.gov/exposurereport/

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Concentrations of Mercury in the Blood of Women of Child-bearing Age (Measure B4)

National Health and Nutrition Examination Survey

Data on mercury levels were obtained from the National Health and Nutrition Examination Survey (NHANES) 1999-2000, conducted by the National Center for Health Statistics. See above for description of NHANES under "Concentrations of Lead in Blood."

Concentrations of Cotinine in Blood (Measure B5)

National Health and Nutrition Examination Survey

Data on children's cotinine levels were obtained from the National Health and Nutrition Examination Survey (NHANES) III and NHANES 1999-2000, conducted by the National Center for Health Statistics. See above for description of NHANES under "Concentrations of Lead in Blood."

Only cotinine levels for nonsmoking children were used. Children were classified as smokers if they had a serum cotinine level greater than 10 nanograms per milliliter (ng/mL).

Respiratory Diseases (Measures D1-D4)

National Health Interview Survey

Data on the prevalence of asthma and other respiratory diseases are from the National Health Interview Survey (NHIS), a continuing nationwide sample survey of the civilian noninstitutionalized population in which data are collected by personal household interviews. Interviewers obtain information on personal and demographic characteristics, including race and ethnicity, by self reporting or as reported by an informant for children under 18 years of age. Investigators also collect data about illnesses, injuries, impairments, chronic conditions, activity limitation caused by chronic conditions, use of health services, and other health topics. For most health topics, the survey collects data over an entire year.

The NHIS sample includes an over-sample of Black and Hispanic persons and is designed to allow the development of national estimates of health conditions, use of health services, and health problems of the U.S. civilian non-institutionalized population. Over the years, the response rate for the ongoing part of the survey has run between 94 and 98 percent. In 2000, interviewers collected information on 32,374 persons 18 years or older, and 13,376 children ages 0-17 years old.

Descriptions of the survey design, the methods used in estimation, and the general qualifications of the data are presented in the following:

- Massey, J.T., T.F. Moore, V.L. Parsons, and W. Tadros. 1989. Design and estimation for the National Health Interview Survey, 1985-1994. Vital and Health Statistics 2 (110). Hyattsville, MD: National Center for Health Statistics.
- Botman S.L., T.F. Moore, C.L. Moriarity, and V.L. Parsons. 2000. Design and estimation for the National Health Interview Survey, 1995-2004. Vital Health Statistics 2 (130). Hyattsville, MD: National Center for Health Statistics.
- Bloom B, and L. Tonthat. 2002. Summary Health Statistics for U.S. Children: National Health Interview Survey, 1997. Vital Health Statistics 10 (203). Hyattsville, MD: National Center for Health Statistics.

Appendix B: Data and Methods

Where possible, the report presents breakouts of the NHIS data for three family income levels: below poverty level; between poverty level and twice the poverty level (shown in graphs and tables as 100-200% of Poverty Level); and greater than twice the poverty level (>200% of Poverty Level). For approximately 15 percent of children represented in the NHIS, family income information needed for this classification was not available. These children are not included in the graphs showing statistics by income level. Statistics for these children are included in the data tables in Appendix A, under "unknown income."

In 1997, the NHIS underwent a major redesign in which the questions used to estimate asthma prevalence were changed. For asthma up to 1996, Measure D1 uses the parent's response to the following question "Did <child's name> have asthma in the past 12 months?" For asthma in 1997-2000, Measures D1 and D2 use parents' response to the following two questions: "Has a doctor or other health professional EVER told you that <child's name> had asthma?" and if yes, "During the past 12 months, has <child's name> had an episode of asthma or an asthma attack?"

In 2001, the NHIS added the following new question: "Does <child's name> still have asthma?" This question was used to estimate the percentage of children who currently have asthma.

NHIS Web site: http://www.cdc.gov/nchs/nhis.htm

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National Hospital Ambulatory Medical Care Survey

Data on asthma emergency room visits were obtained from the National Hospital Ambulatory Medical Care Survey (NHAMCS). The NHAMCS is designed to collect data on ambulatory care services in hospital emergency and outpatient departments. Findings are based on a national sample of visits to the emergency departments and outpatient departments of noninstitutional general and short-stay hospitals, exclusive of federal, military, and Veterans Administration hospitals, located in the 50 states and the District of Columbia. Annual data collection began in 1992.

Specially trained interviewers visit the hospitals prior to their participation in the survey to explain survey procedures, verify eligibility, develop a sampling plan, and train hospital staff in data collection procedures. Hospital staff are instructed to complete patient record forms for a systematic random sample of patient visits during a randomly assigned four-week reporting period. Data are obtained on demographic characteristics of patients; expected source(s) of payment; patients' complaints; physicians' diagnoses; diagnostic and screening services; procedures; medication therapy; disposition; types of health care professionals seen; causes of injury where applicable; and certain characteristics of the hospital, such as the type of ownership.

Respiratory infections are ICD-9 codes 464 and 465, acute bronchitis is ICD-9 code 466, and asthma is ICD-9 code 493.

NHAMCS Web site: http://www.cdc.gov/nchs/about/major/ahcd/ahcd1.htm

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National Hospital Discharge Survey

Data on asthma hospitalizations were obtained from the National Hospital Discharge Survey (NHDS). The NHDS is a national probability survey designed to meet the need for information on characteristics of inpatients discharged from non-federal short-stay hospitals in the United States. The NHDS collects data from a sample of approximately 270,000 inpatient records acquired from a national sample of approximately 500 hospitals. Only hospitals with an average length of stay of fewer than 30 days for all patients, general hospitals, or children's general hospitals are included in the survey. Federal, military, and Department of Veterans Affairs hospitals, as well as hospital units of institutions (such as prison hospitals), and hospitals with fewer than six beds staffed for patient use, are excluded. Data from the NHDS are available annually.

Respiratory infections are ICD-9 codes 464 and 465, acute bronchitis is ICD-9 code 466, and asthma is ICD-9 code 493.

NHDS Web site: http://www.cdc.gov/nchs/about/major/hdasd/nhdsdes.htm

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Childhood Cancer (Measures D5-D6)

Surveillance, Epidemiology, and End Results Program

The population-based data used for incidence of cancer are from the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute (NCI). Information from five states (Connecticut, Hawaii, Iowa, New Mexico, and Utah) and five metropolitan areas (Atlanta, Georgia; Detroit, Michigan; Los Angeles, California; San Francisco-Oakland, California; and Seattle-Puget Sound, Washington) accounting for approximately 14 percent of the United States' population are included. The participating regions were selected primarily for their ability to operate and maintain a population-based cancer reporting system and for their epidemiologically significant population subgroups. With respect to selected demographic and epidemiologic factors, they are, when combined, a reasonably representative subset of the U.S. population.

The mortality data for all cancer deaths among children in the United States are from data based on underlying cause of death from the National Vital Statistics System, administered by the National Center for Health Statistics. Mortality data are obtained by NCI and provided for all causes of cancer.

All rates are age-adjusted to the 1970 U.S. standard population.

SEER Web site: http://seer.cancer.gov

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Appendix B: Data and Methods

Neurodevelopmental Disorders (Measure D7)

National Health Interview Survey

Data on the prevalence of attention-deficit/hyperactivity disorder and mental retardation are from the National Health Interview Survey (NHIS). See above for description of the NHIS under "Respiratory Measures." For mental retardation, the measure uses parents' responses to this question: "Has a doctor or health professional ever told you that <child's name> had mental retardation?" For attention-deficit/hyperactivity disorder, the measure uses parents' responses to this question: "Has a doctor or health professional ever told you that <child's name> had attention deficit disorder?"

Advisories for Methylmercury in Non-Commercial Fish

National Listing of Fish and Wildlife Advisories

EPA's Office of Water maintains a database that includes all advisories issued by states, territories, and tribes to warn people to reduce or eliminate consumption of fish due to chemical contamination. Most of the advisories are for non-commercial fish that people catch for their own use, but some advisories issued by states for commercial fish also are included. Some of the advisories apply to entire states; others apply only to specified water bodies such as lakes or rivers. This database can be searched by state, by type of fish, or by contaminant. The database was used for Measure E9, which reports the number of women of child-bearing age living in states that have issued advisories for mercury in non-commercial fish. The database includes advisories issued through the year 2000. A fact sheet describing the database is available at http://www.epa.gov/ost/fish/advisories/factsheet.pdf.

The Office of Water also has produced extensive guidance to assist states and tribes in developing and issuing advisories. These guidance documents are available at http://www.epa.gov/ostwater/fish/guidance.html.

The National Listing of Fish and Wildlife Advisories database is available at:
http://map1.epa.gov/

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Lead in California Public Elementary Schools and Childcare Facilities (Measures S1-S3)

California Department of Health Services Study on Lead Contamination in California Schools

After the California state legislature passed the Lead-Safe Schools Protection Act in 1992, the Childhood Lead Poisoning Prevention Branch of the California Department of Health Services commissioned a study to determine the extent of lead contamination in paint, soil and water in California schools. The study began in 1994 and the collection of data was completed in 1997.

The Department of Health Services randomly selected 200 of the 5,041 public elementary schools in California to participate in the study. The participating schools were selected to reflect the statewide distribution of school buildings by age (the age of the building is a predictor of the presence of lead). The selection process also yielded a geographically representative sample. Schools with both public and private pre-kindergarten and childcare programs located on their premises were included in the sample.

Trained field staff collected the samples and an accredited commercial laboratory conducted the analysis. Samples in all media were analyzed using Flame Atomic Absorption Spectroscopy. Paint and soil samples were taken from the oldest building in the school and the youngest children's classroom. In 76.5 percent of the schools, the oldest building also housed the youngest children's classroom. Water samples also were taken from the oldest building except in cases in which there was no water outlet in the building.

Paint chip samples were taken from walls, doors, or windows. Wherever possible, they were obtained from areas where the paint was visibly deteriorated. Soil samples were collected from within five feet of painted walls or windows, from play areas, and from a location on the school grounds that was as far away from any building as possible. Water samples were taken from outlets located both inside and outside the school buildings.

More information on study design, methods, and results is available at
http://www.dhs.ca.gov/childlead/schools/sitemap.htm

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Minnesota Survey on Pesticide Use in K-12 Schools

Pesticide Use in Minnesota's K-12 Schools (Measure S4)

In 1998, the Minnesota Department of Agriculture and the University of Minnesota jointly established an Integrated Pest Management (IPM) workgroup in response to growing concerns about pesticide use in K-12 schools in the state. The workgroup included representatives from both founding organizations; the Minnesota Office of Environmental Assistance; the Minnesota Department of Children, Families and Learning; the Minnesota Department of Health; and the St. Paul Public Schools.

In 1999, the workgroup commissioned a statewide survey of pest management practices in K-12 schools, funded by EPA. The purpose of the survey was to obtain statistically reliable statewide data that would provide general information about current pest management practices in K-12 schools.

The survey questionnaires were designed by the workgroup and the survey was formatted and conducted by an independent contractor, C.J. Olson Market Research, Inc. A pretest was mailed out to 25 superintendents and 25 head custodians (those who received the pre-tests were not contacted again). The final survey questionnaires were mailed to 330 superintendents out of the 355 districts in the state, and to a random sample of 1,160 public and private K-12 schools out of 2,197 school buildings in the state. The questionnaires mailed to the schools were addressed to principals, who were instructed to ask head custodians to complete the questionnaires.

Response to the questionnaires was voluntary. The overall response rate for the entire survey was 36 percent. One hundred and sixty eight (168) superintendents' questionnaires and 375 head custodians' questionnaires were processed. The responses from the head custodians were used for this report.

More information about the survey is available at
http://www.mda.state.mn.us/IPM/PestMgmtinSchools.html

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California Birth Defects Monitoring Program

Birth Defects (Measure S5)

The California Birth Defects Monitoring Program registry is a population-based registry for babies born in the following counties in California: Fresno, Kern, Kings, Los Angeles, Madera, Merced, San Francisco, San Joaquin, Santa Clara, Stanislaus, and Tulare. These counties represent about 45 percent of the state population. Experience in monitoring California rates and trends since 1983 shows that the rates from the eight Central Valley counties are reflective of those throughout California: both urban and rural areas are included, all major racial/ethnic groups are present in the birth population in similar proportions to what is seen in California as a whole, and the high quality of diagnostic practices in the primary hospitals and referral centers yield thorough and accurate case identification in medical records. This sampling of California's birth population is the same as that used by the Centers for Disease Control and Prevention for the National Birth Defects Prevention Study.

Babies born with birth defects serious enough to require medical treatment or to cause disability are included in the registry. These birth defects are listed in codes 740-759 of the International Classification of Diseases (ICD) and include structural birth defects such as missing limbs and malformed organs, chromosome abnormalities such as Down syndrome, and birth defects patterns such as fetal alcohol syndrome. The cases are ascertained actively through data gathering from medical facilities and review of medical records. Birth defects diagnoses made prenatally, after birth, and up to one year of age are included in the registry.

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Child Population Data

U.S. Census County-Level Data

County population estimates were obtained from the U.S. Census Bureau. For 1990, data from the decennial census were used directly. For subsequent years, county population data were estimated by the Census Bureau using information on births, deaths, domestic migration, and international migration. Individual age populations were compiled for children ages 0 to 17 at the county level for each year 1990 to 1999. A complete description of the population estimation methodology can be found on the Census Bureau's Methodology for Estimates of State and County Total Population Web page at http://www.census.gov/population/methods/stco99.txt and on the Census Bureau's Methodology for Estimating County Population by Age and Race Web site at http://www.census.gov/population/estimates/county/casrh_doc.txt.

The U.S. Census Bureau population estimates for 1990 to 1999 classify the population among six race/ethnic categories, which herein are referred to as the unadjusted race categories. These categories are White non-Hispanic, White Hispanic, total Black (Hispanic and non-Hispanic), total American Indian (Hispanic and non-Hispanic), and total Asian and Pacific Islander (all races with Hispanic origin indicated). U.S. Census data were adjusted to reflect five separate race/ethnic categories that were used in this report: White non-Hispanic, Black non-Hispanic, American Indian and Alaska Native non-Hispanic, Asian and Pacific Islander non-Hispanic, and Hispanic.

For the 2000 census, individuals were allowed to report two or more race groups when responding to the census. As with the 1990 census, individuals may indicate whether they are of Hispanic origin in addition to indicating their race. Persons reporting multiple races in the 2000 data were assigned to one of the five race/ethnicity categories used in this report (Black non-Hispanic, White non-Hispanic, American Indian and Alaska Native Non-Hispanic, Asian and Pacific Islander non-Hispanic, and Hispanic) using the method described below.

Census data also were used to derive children's population counts for three income categories: 1) households with income below poverty level; 2) households with income greater than or equal to poverty level but less than twice the poverty level; and 3) households with income equal to or greater than 200 percent of poverty level. The Census Bureau defines poverty level based on a set of money income thresholds that vary by family size and composition. If a family's total income is less than that family's threshold, then that family, and every individual in it, is considered poor. The Census

Bureau updates its poverty thresholds annually. In 2000, a family of two adults and two children with total income below $17,463 was considered below the poverty level. Tables showing the Census Bureau's poverty thresholds are available at http://www.census.gov/hhes/poverty/threshld.html.

Agency Contact:
U.S. Census Bureau
Population Estimates Branch
(301) 457-2385
http://www.census.gov/population/www/estimates/countypop.html

Methods for Race Adjustment for 1990-1999 County Census Data

The overlapping race and ethnicity categories were adjusted into the non-overlapping categories of White non-Hispanic, Black non-Hispanic, American Indian and Alaska Native (AIAN) non-Hispanic, Asian and Pacific Islander (API) non-Hispanic, and Hispanic, for children under the age of 18. Census data provide 1990-1999 population estimates by "Race by Hispanic origin" (e.g., percentage of Black Hispanic and Black non-Hispanic) but do not provide these data by sex or age, except for White Hispanics and White non-Hispanics. An Hispanic ethnicity correction factor, consisting of the ratio of Black non-Hispanics to total Blacks, was calculated for all Blacks. This ratio was multiplied by the total number of Black children to estimate the number of Black non-Hispanic children. A similar procedure was followed for AIAN and API children. The analysis assumed that the ratio of Hispanic to non-Hispanic children in each race category was the same as the corresponding ratio for the total population. The Hispanic ethnicity correction factors for Black, AIAN, and API were applied to the population of children on a county-by-county basis. This method created five mutually exclusive race/ethnicity categories: Black non-Hispanic, White non-Hispanic, AIAN non-Hispanic, API non-Hispanic, and Hispanic, for children 17 and under.

Methods for Race Adjustment for 2000 County Census Data

Starting in 2000, persons can report multiple races to describe their race on the census form. This analysis uses the bridging methodology by Parker and Makuc[1] for assigning non-Hispanic persons who report multiple race categories to four mutually exclusive race categories: White non-Hispanic, Black non-Hispanic, AIAN non-Hispanic, and API non-Hispanic. The Parker and Makuc methodology is based on race information collected from the 1993-1995 National Health Interview Survey. In this survey, respondents who reported multi-racial categories also were asked to report a single race with which they would identify themselves. Parker and Makuc calculated proportions of multi-racial respondents who would have identified themselves with a single race if that category were the only option. Major bi-racial categories identified by the authors were: White/Black, White/AIAN, White/API, and Black/AIAN. In this analysis, equations were derived for apportioning these four bi-racial categories into single-race categories using the proportions reported by Parker and Makuc. Race combinations other than the major bi-racial groups identified by Parker and Makuc were grouped under the category of "multiple race." The multiple race category was further split into the four single-race categories using the national distribution of the four single-race categories in the United States (e.g., 71.3 percent of "multiple race" respondents were considered White).

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[1] Parker, J.D. and Makuc, D.M. 2002. Methodological implications of allocating multiple race data to single race categories. *Health Services Research* 37 (1): 203-15.

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Methods for Obtaining Counts of Children under Age 18 by Race/Ethnicity

The Census Bureau provides county-specific population estimates by race and ethnicity for each year from 1990-1999 for several age groups, including ages 15-19. For the measures in this report, which are focused on children under age 18, it was therefore necessary to estimate the portion of the population age 15-19 that is under age 18 (i.e., ages 15-17). Census Bureau files on population by county provide estimates of people for each age (i.e., 15 year olds, 16 year olds, etc.) for each year. A scaling factor was calculated for each county, for each year, as the proportion of 15-19 year olds who are ages 15-17. This scaling factor was multiplied by the estimated population ages 15-19 for each race/ethnicity to estimate the number of children ages 15-17, by race/ethnicity, for each county for 1990- 1999. This calculation assumes that the proportion of 15-19 year olds that is 15-17 years old is constant across the race/ethnicity categories in each county.

Methods for Calculating Population by Ratio of Income to Poverty Level

Population counts by the three categories of income to poverty level ratio (households with income below poverty level, households with income greater than or equal to poverty level but less than twice the poverty level, and households with income equal to or greater than 200 percent of poverty level) are available for the 1990 census but are not estimated by the Census Bureau for the intercensal years 1991 to 1999. For the intercensal years, the Census Bureau's county-level children's population estimates were multiplied by the percentage of children in a given poverty range from the 1990 population data to estimate the number of children, by county, in the three income categories.

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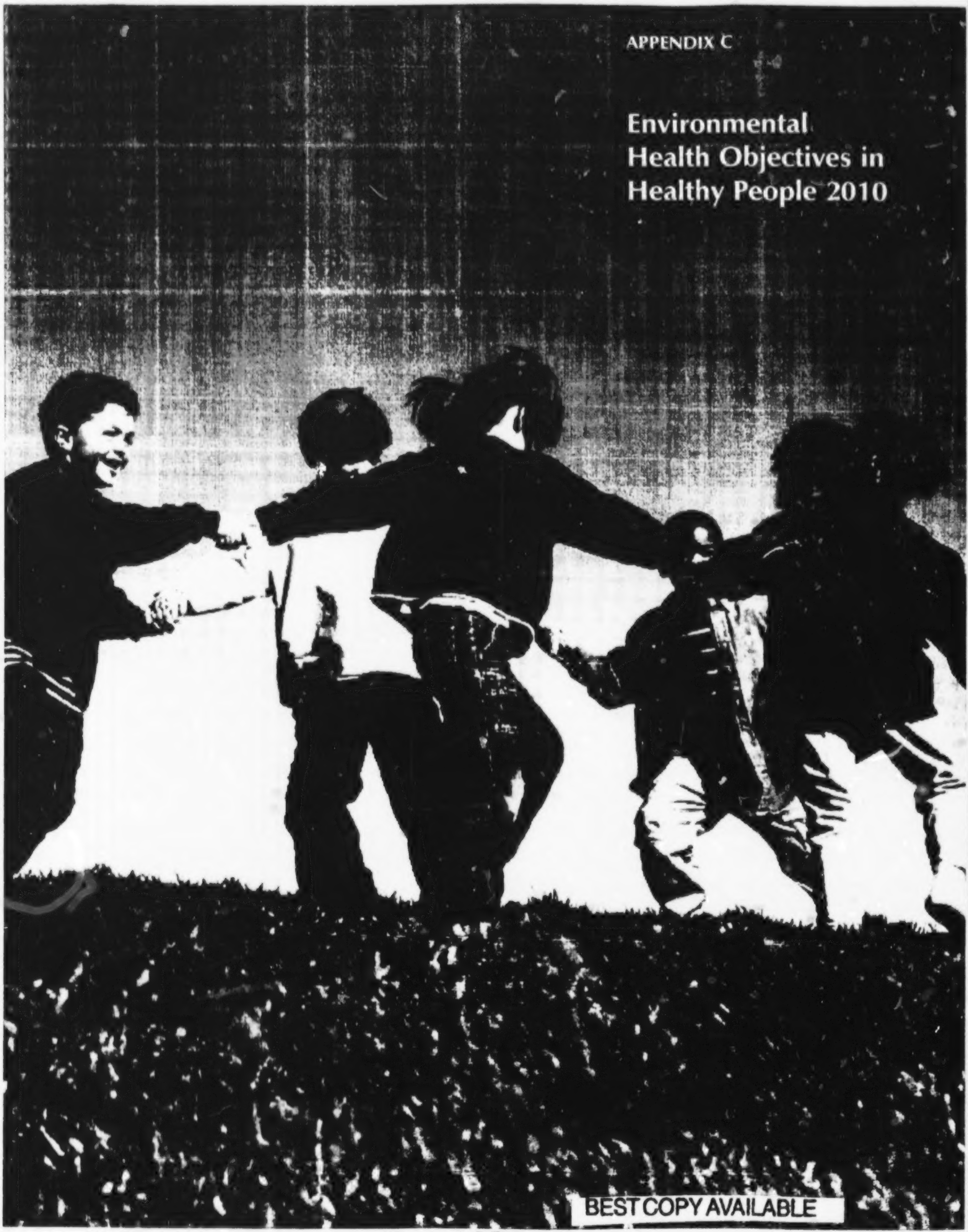
APPENDIX C

Environmental Health Objectives in Healthy People 2010



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Appendix C: Environmental Health Objectives in Healthy People 2010

Healthy People 2010, coordinated by the U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion, establishes national health objectives for the first decade of the new millennium. Launched in January 2000, Healthy People 2010 seeks to increase the quality and number of years of healthy life and to eliminate health disparities among Americans.

Healthy People 2010 includes a number of goals and objectives that relate to the children's environmental health risks considered in *America's Children and the Environment.*

Objective 8-1 is to reduce the proportion of persons exposed to air that does not meet the U.S. Environmental Protection Agency's health-based standards for harmful air pollutants.

Objective 8-4 is to reduce air toxic emissions to decrease the risk of adverse health effects caused by airborne toxics.

Objective 8-5 is to increase the proportion of persons served by community water systems who receive a supply of drinking water that meets the regulations of the Safe Drinking Water Act.

Objective 8-10, currently under development, is to reduce the potential human exposure to persistent chemicals by decreasing fish contaminant levels.

Objective 8-11 is to eliminate elevated blood lead levels in children.

Objective 8-12 is to minimize the risks to human health and the environment posed by hazardous sites.

Objective 8-24 is to reduce exposure to pesticides as measured by urine concentrations of metabolites.

Objective 16-14 is to reduce the occurrence of developmental disabilities.

Objective 24-2a is to reduce hospitalizations for asthma for children under 5.

Objective 27-9 is to reduce the proportion of children who are regularly exposed to tobacco smoke at home.

Healthy People 2010 is available at www.health.gov/healthypeople or by calling 1(800) 367-4725.

APPENDIX D

Environmental Health Objectives in EPA's Strategic Plan

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Appendix D: Environmental Health Objectives in EPA's Strategic Plan

Relevant Environmental Health Objectives in EPA's 2000-2005 Strategic Plan

EPA's mission is to protect human health and to safeguard the natural environment—air, water, and land—upon which life depends. The agency's 2000-2005 Strategic Plan lays out long-term goals and shorter-term objectives for fulfilling that mission.

The plan's 10 long-term goals establish EPA's major priorities for the five-year period. These include 1) clean air; 2) clean and safe water; 3) safe food; 4) preventing pollution and reducing risk in communities, homes, workplaces, and ecosystems; 5) better waste management, restoration of contaminated waste sites, and emergency response; 6) reduction of global and cross-border environmental risks; 7) quality environmental information; 8) sound science, improved understanding of environmental risk, and greater innovation to address environmental problems; 9) a credible deterrent to pollution and greater compliance with the law; and 10) effective management.

A number of objectives in the plan relate to the risks to children's environmental health considered in *America's Children and the Environment.* Those selected objectives are presented here.

The full text of EPA's 2000-2005 Strategic Plan is available at http://www.epa.gov/ocfo/plan/2000strategicplan.pdf.

Goal 1: Clean Air

Objectives:

- Reduce the risk to human health and the environment by protecting and improving air quality so that air throughout the country meets national clean air standards by 2005 for carbon monoxide, sulfur dioxide, nitrogen dioxide, and lead; by 2012 for ozone; and by 2018 for particulate matter.
- By 2020, eliminate unacceptable risks of cancer and other significant health problems from air toxic emissions for at least 95 percent of the population, with particular attention to children and other sensitive subpopulations, and substantially reduce or eliminate adverse effects on our natural environment.

Goal 2: Clean and Safe Water

Objectives:

- By 2005, protect human health so that 95 percent of the population served by community water systems will receive water that meets health-based drinking water standards, consumption of contaminated fish and shellfish will be reduced, and exposure to microbial and other forms of contamination in waters used for recreation will be reduced.
- By 2005, reduce pollutant loadings from key point and nonpoint sources by at least 11 percent from 1992 levels. Air deposition of key pollutants will be reduced to 1990 levels.

Goal 3: Safe Food

Objectives:

- By 2006, reduce public health risk from pesticide residues in food from pre-Food Quality Protection Act levels (pre-1996).
- By 2008, use on food of current pesticides that do not meet the new statutory standard of "reasonable certainty of no harm" will be eliminated.

Goal 4: Preventing Pollution and Reducing Risk in Communities, Homes, Workplaces, and Ecosystems

Objectives:

- By 2005, public and ecosystem risk from pesticides will be reduced through migration to lower-risk pesticides and pesticide management practices, improving education of the public and at-risk workers, and forming "pesticide environmental partnerships" with pesticide user groups.
- By 2007, significantly reduce the incidence of childhood lead poisoning and reduce risks associated with polychlorinated biphenyls (PCBs), mercury, dioxin, and other toxic chemicals of national concern.
- By 2005, 16 million more Americans than in 1994 will live or work in homes, schools, or office buildings, with healthier indoor air.
- By 2005, facilitate the prevention, reduction, and recycling of toxic chemicals and municipal solid wastes, including PBTs. In particular, reduce by 20 percent the actual (from 1992 levels) and by 30 percent the production-adjusted (from 1998 levels) quantity of Toxic Release Inventory-reported toxic pollutants that are released, disposed of, treated, or combusted for energy recovery, half through source reduction.

Goal 5: Better Waste Management, Restoration of Contaminated Waste Sites, and Emergency Response

Objectives:

- By 2005, EPA and its federal, state, tribal, and local partners will reduce or control the risk to human health and the environment at more than 374,000 contaminated Superfund, RCRA, underground storage tank (UST), and brownfield sites and have the planning and preparedness capabilities to respond successfully to all known emergencies to reduce the risk to human health and the environment. (Total comprises 1,105 NPL sites, 1714 RCRA facilities, 370,000 UST cleanups initiated or completed, and 1,500 brownfield properties.)
- By 2005, EPA and its federal, state, tribal, and local partners will ensure that more than 277,000 facilities are managed according to the practices that prevent releases to the environment. (Total comprises 6,500 RCRA hazardous waste treatment, storage, and disposal facilities, and municipal solid waste landfills; 264,000 USTs, and 7,100 oil facilities.)

Goal 7: Quality Environmental Information

Objectives:

- Through 2006, EPA will continue to increase the availability of quality health and environmental information through educational services, partnerships, and other methods designed to meet EPA's major data needs, make data sets more compatible, make reporting and exchange methods more efficient, and foster informed decision making.
- By 2006, EPA will provide access to new analytical or interpretive tools beyond 2000 levels so that the public can more easily and accurately use and interpret environmental information.
- Through 2006, EPA will continue to improve the reliability, capability, and security of EPA's information infrastructure.

U.S. Department of Education
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